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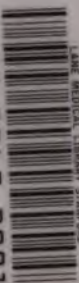
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*Vide page 302.*

CLINICAL LECTURES  
ON  
DISEASES OF THE HEART  
AND AORTA

BY  
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## PREFACE TO THE FIRST EDITION.

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“I hold every man a debtor to his profession; from the which as men of course do seek to receive countenance and profit, so ought they of duty to endeavour themselves, by way of amends, to be a help and ornament thereunto.”—*The Works of Francis Bacon*, London, 1859, vol. vii. p. 319.

WITH Latham I may say that mine is but “a limited purpose—it is to regard the diseases of the heart only in one point of view, *i.e.*, as they appear in the living man.” As this is the object of clinical teaching, I have called these lectures clinical, though otherwise the term is somewhat of a misnomer, as the illustrations have been mainly taken from patients who have passed from the clinique into the domain of morbid anatomy. One obvious advantage of this method is the definite connection, provided the cases are sufficiently numerous, of certain distinct *pre-mortem* phenomena with equally well-defined *post-mortem* appearances; a sufficient guarantee for the accuracy of this connection being to be found in the publicity inseparable from a large teaching hospital like the Edinburgh Infirmary, and also in the fact that the life history is closed by the physician in the case-book before the pathologist commences his record of the morbid appearances. In a work such as the present it was impossible to avoid narrating cases, but I have restricted these to as few as possible, giving only the histories of those patients who may be regarded as affording well-marked examples of the phenomena attending any given lesion.

No two cases of any disease are ever exactly alike, even though depending upon the same central lesion. The object of all clinical teaching is to show how we are led to determine

what is the central lesion in any given case, and to explain how the apparently inexplicable phenomena, which constitute what we term the disease, group themselves naturally round that lesion in accordance with physical and physiological laws; and finally, to show how we can influence that central lesion by remedies, and how effectually this modification of the central cause modifies also the concomitant phenomena, though now and then some of these become so developed as to require special and independent treatment, a treatment which can never be anything but palliative, while that of the central lesion may not infrequently be really curative. All this I have endeavoured to do in relation to cardiac disease, and though none of the lectures have ever been actually delivered as they now stand, they yet comprise the essence of my clinical teaching, in regard to diseases of the heart and aorta, during the last eight years. By putting this into the lecture form, I have been enabled to write with more ease to myself, and, what is of more importance, I have been enabled to avoid noticing any special subject which has not happened to come under my own observation. I have thus been enabled to avoid the mere repetition of the statements of those who have preceded me in the same field of inquiry, and by transcribing my descriptions of disease directly from the book of nature, and basing my explanations of the phenomena observed—and, to a large and most important extent, their treatment also—upon physical laws and the results of experimental physiology.\* I have done what I could to remove

\* I may as well state here that, though I myself never performed one single experiment upon a living animal, and though I deprecate as much as any one the undue multiplication of such experiments, I yet hold it to be true that much that is now plain in cardiac diagnosis, and definite in the treatment of cardiac disease, has been made so by means of experimental physiology, and that many patients now living owe their lives primarily to this method of investigation, a method of investigation which, kept within due bounds, cannot be regarded as inconsistent with Christian humanity, when we reflect that to save one life alone a whole herd of swine were sent into the depths of the sea of Gennesareth, and that we are also told "ye are of more value than many sparrows," or frogs either.

the diagnosis and treatment of these diseases from the domain of mere speculative opinion, and to place them under direct scientific control. And if in any respect I have failed in doing so, the failure is undoubtedly due to my own ignorance, and will by and by be remedied by some worthier successor, who will base his diagnosis upon a more successful application of the laws and facts of physics, to the explanation of the phenomena connected with cardiac disease, and rest his treatment upon more accurate physiological knowledge. For "the thoughts of men are widened by the process of the suns," and to trace this process in the diagnosis and treatment of cardiac disease we neither require to go very far back in medical history, nor even to read many books. For though in 1531 the pulmonary circulation was shown by Servetus to be reasonably probable,\* yet it was not till 1628†—nearly a century afterwards—that Harvey first gave to the world a full account of the double systemic and pulmonary circulation, which he had already publicly taught and demonstrated since 1619. Of course, any attempts at the diagnosis of cardiac diseases previous to this period can possess no scientific value whatever, and even their treatment must have been founded on the vaguest empiricism. And for long after this era, as before it, the various essays upon diseases of the heart are of no value except in so far as they contain a number of curious pathological observations. Even Senac's great work, the first complete monograph upon diseases of the heart, is solely valuable in this respect.‡ Thus, while Harvey may be considered to have founded the modern physiology of the heart and the circulation in 1628, and Morgagni must be equally regarded as the founder of the modern pathology of the organs concerned in 1762,§ it is from Corvisart that we

\* *De Trinitatis Erroribus*. Basil, 1531.

† *Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus* Francoferæ, 1628.

‡ *Traité de la Structure du Cœur, de son Action, et de ses Maladies*. Paris, 1749.

§ *De Sedibus et Causis Morborum per Anatomea indagatis*. Venet. 1762.

date the origin of the modern system of diagnosis—in 1806.\* Corvisart was first physician to the Great Napoleon, and was said to have been the only man of independent mind about his court, having been not more remarkable for his modesty than for his skill, especially in diagnosis. As a sample of the high estimation in which his diagnostic powers were held both by the laity and the profession, two anecdotes may be related. In one he is represented as standing before a portrait and saying, "If the artist painted this picture correctly from the life, the original of this portrait must be already dead from disease of the heart." It is added that this was actually the case. In the other, Dupuytren is represented as saying, "I have often observed the marvellous skill with which Corvisart not only pointed out the nature and position of a cardiac lesion, and indicated almost to a line the exact size of that cardiac orifice which he had diagnosed as contracted." We know very well that no amount of diagnostic skill would, even now-a-days, justify either statement, though by a species of *Εύστοχία* Corvisart might by chance have been right enough in occasional instances. He was the first to make practical use of Auenbrugger's† invention (1761) of percussion, and he was also in the habit of listening to the sounds made by the heart, but it was to Laennec,‡ as every one knows, that we owe (1819) the first attempt at the scientific application of auscultation to the diagnosis of disease. And it is to Bouillaud§ (1824) that we owe the first application of scientific auscultation to the diagnosis of cardiac disease, thus opening up the means of attaining an accuracy, the possibilities of which are as yet only dawning on the professional mind. But it is to the clinical experience, and above all, to the practical

\* *Essai sur les Maladies et les Lésions Organique du Cœur et des gros Vaisseaux.* Paris, 1806.

† *Inventum Novum ex Percussione Thoracis humani, ut signo abstrusos pectoris morbos detegendi.* Vindobonæ, 1761.

‡ *De L'Auscultation médiate.* Paris, 1819.

§ *Traité Clinique des Maladies du Cœur.* Paris, 1824.



experiments of James Hope \* (1832), that we owe a very considerable advance in our knowledge of the causes of cardiac murmurs, in particular, I believe he is the first who noted the occurrence of mitral regurgitation from simple dilatation of the ventricle, the orifice being healthy. To Bouillaud, however, following Laennec, we owe the distinct statement that the auricle is found to pulsate alternately with the ventricles in certain cases of disease, particularly in cases of mitral constriction. And this observation, which is altogether slighted by Hope, had no doubt its influence in leading M. Forget † (1851) to his doctrine of retro-dilatation, which is the first distinct, though very imperfect, attempt at the foundation of a physiologico-pathology of the morbid heart, ‡ a mode of investigation which seems destined to produce very important results in the detection of cardiac diseases. Closely following Forget, we have the very instructive work of Stokes § (1854), whose interesting chapters upon the condition of the heart in fevers, and upon the effect of defective cardiac power upon the cerebral circulation, are amongst the most important additions made in recent times to our knowledge of cardiac pathology. The only other work that I have to add to this list, fully to bring down the history of cardiac diagnosis to the present day, is the admirable compendium of Von Dusch || (1868), which has, in my opinion, no equal in any language as a practical exposition of the art of diagnosing diseases of the heart.

It is not yet seventy years since science in any form became applied to the diagnosis of cardiac disease; each of the works which I have enumerated constitutes an era in the history of the development of this diagnosis, and a consideration of the half dozen works I have recorded is sufficient to enable us to

\* *Treatise on the Diseases of the Heart and Great Vessels.* London, 1832.

† *Précis Théorique et Pratique des Maladies du Cœur.* Strasbourg et Paris, 1851.

‡ *Op. cit.* p. 18.

§ *The Diseases of the Heart and Aorta.* Dublin, 1854.

|| *Lehrbuch der Herzkrankheiten.* Leipzig, 1868.

trace the progress of cardiac diagnosis from its earliest dawnings quite down to the present day. To mark more accurately the gradual yet decided advance, I have purposely quoted the first edition of each work; for the views first promulgated by Bouillaud and Hope, for instance, were very much modified in subsequent editions, and the student loses very much the sensation of gradual progress if he reads only the last edition of either. In this brief survey of the progress of cardiac diagnosis I have purposely restricted myself to practical treatises of diseases of the heart. To extend the survey wider would be to give an entire bibliography of the subject, which seems to me quite unnecessary. I may, however, add that the only two modern works on percussion and auscultation which contain important novelties in the practical application, or the scientific explanation of these arts, are those of Skoda \* and of Paul Niemeyer.† And I may also mention the work of Allan Burns ‡ as containing some important observations, especially in regard to mitral stenosis and epigastric pulsation; the paper by Billing§ on the sounds of the heart, as containing the first promulgation of views in regard to their production, which have since been adopted, and upon which much of modern diagnosis hangs, and in this respect Rouanet's|| paper is also of importance; also Corrigan's¶ important paper on aortic regurgitation; and the papers by Fauvel,\*\* Gairdner,†† Hilton Fagge,‡‡ and Galabin,§§ upon mitral stenosis.

In regard to the treatment of cardiac disease, it is only in

\* *Abhandlung über Perkussion und Auscultation.* Wien, 1839.

† *Theoretischen und Clinischen Handbuch der Perkussion und Auscultation.* Erlangen, 1870.

‡ *On some Diseases of the Heart.* Edinburgh, 1809.

§ *Lancet*, May 1832.

|| *Journal Hebdomadaire*, No. 97.

¶ *Edinburgh Medical and Surgical Journal*, vol. xxxvii. 1832.

\*\* "Memoire sur les signes Stethoscopiques du Rétrécissement de l'orifice auriculo-ventriculaire gauche du Cœur," *Archives Générales de Médecine*, tom. i. 1843.

†† *Edin. Med. Jour.* Nov. 1861.

‡‡ *Guy's Hospital Reports*, 3d series, vol. xvi.

§§ *Ibid.* vol. xx.

recent times that any important advance has been made. Senac says: "A mesure qu'on pénètre dans les maladies du cœur, la médecine paraît plus stérile; que peut-on espérer des médicaments, par exemple, dans les dilatations du cœur?" \* But to this doleful query modern medicine has given a most triumphant reply, and can truly say that there are few diseases more amenable to treatment than moderate dilatation of the heart, while every form of cardiac disease is susceptible of a degree of relief which but a few years ago was wholly undreamed of; but for a further consideration of this I must refer to the body of this work generally, and particularly to chaps. iii., vi., vii., xii., and xiv.; and I believe that a comparison of these chapters with even the latest works on cardiac therapeutics will show that a very considerable advance in this respect has been made in quite recent times, and that this advance is undoubtedly due, or at all events owes much of its precision, to the researches of physiologists. I regret very much that I can give no definite statistical information as to the prevalence of the various forms of disease of the heart, because my own time and attention have been otherwise occupied, and I find that those statistics, accumulated for me by my various resident physicians, are not of much practical importance. During the eight years that I have been connected with the Royal Infirmary here, I have had under my care in its wards considerably over 2000 cases of general disease, exclusive of cases of continued and eruptive fevers. Some of the ward journals have gone amissing, but I have records of 1968 cases, of whom over 200 were cases of cardiac disease. Of these 67 are recorded under the head of aortic regurgitation alone, and 18 under that of aortic and mitral disease; there were 85 therefore in whom aortic regurgitation was the most important lesion. 77 cases are recorded under the head of mitral stenosis, and 48 simply under that of mitral disease; of these, therefore, a presystolic murmur must have

\* *Op. cit.* vol. ii. livre iv. chap. iv. p. 328.

constituted the predominant sign in the first series, and a systolic murmur in the second—125 cases in all of mitral disease, and 213 in all of aortic and mitral disease, exclusive of cases of simple dilatation, of dilated hypertrophy, of tricuspid regurgitation or obstruction, or of pulmonary obstruction. These statistics are obviously of no value except as affording an approximate indication of the numbers of cardiac cases coming annually under my observation in the Infirmary alone. In future I shall endeavour to have the cases of cardiac disease coming under my own care more accurately recorded, both as to their exact nature and as to their probable causation.

I have also to apologise for the large number of cases recorded in the chapter on the treatment of aortic aneurism, as well as for their fulness of detail, but the subject appeared to me of so much importance as imperatively to call for full information. I may add, that I have had under my care in the Infirmary during the last eight years over thirty cases of aortic aneurism, of whom 31 were cases of thoracic aneurism, 23 of them males and 8 females, and 5 were cases of abdominal aneurism (aortic), all males.

In conclusion, I may say, that no one can be more conscious of the many shortcomings of this work than I am myself, or I may add more desirous that any opinions advanced in it should only be received in so far as they shall be found to be consistent with the truths of those sciences upon which the successful practice of our profession is based.

17 WALKER STREET,  
EDINBURGH, *December 1875.*

## PREFACE TO THE SECOND EDITION.

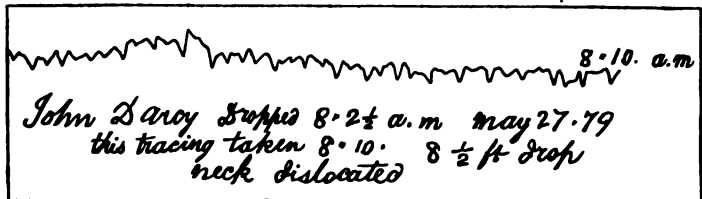
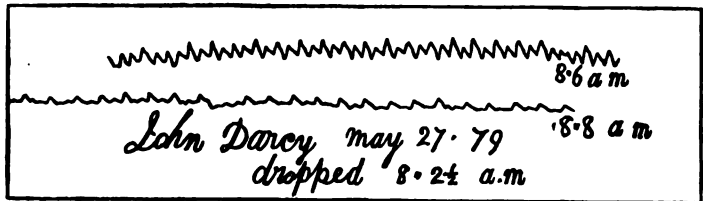
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I HAVE to thank the profession for the kindly manner with which the first edition of this work was received, notwithstanding its many faults. The present edition has been almost entirely re-written and is somewhat enlarged, advantage having been taken of this opportunity to obviate certain misconceptions to which some of the statements in the former edition seem to have been exposed. The living cases formerly narrated have been brought down to date, one or two having fallen out of sight. An attempt has also been made to explain the action and to formulate rules for the use of digitalis in disease of the heart, and of iodide of potassium in aneurism, which, if not absolutely accurate, are at least in accordance with the results obtained by pharmacologists, and will, it is hoped, prove useful to practitioners. As formerly, I desire all my statements of facts, or what I believe to be facts, to be carefully tested; but as no human work is perfect, I claim indulgence for the manner of stating.

G. W. B.

17 WALKER STREET,  
EDINBURGH, *December* 1881.

The two following sphygmographic pulse tracings belong to the curiosities of medical science, and are yet not without a practical interest. I owe them to the kindness of Dr Sidey, the surgeon of our prison, who was the first to observe that after somatic death by hanging, the pulse did not immediately cease, but gradually died away during a period of about twenty minutes. The tracings were taken by Dr Tempest Anderson, surgeon to the jail at York, through whose kind permission, as well as that of Captain Twyford, governor of York Castle, they are here inserted.



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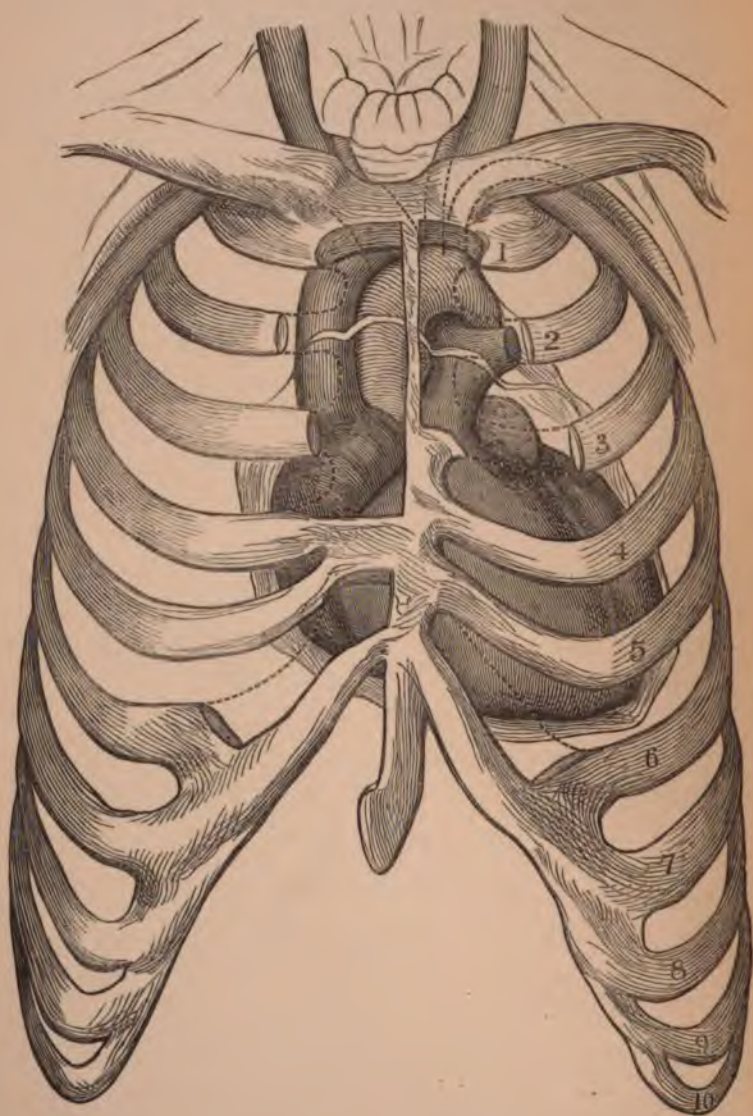
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Slightly altered from Sibson. (*Medical Anatomy*, Plate XIX.)

# CLINICAL LECTURES

ON

## DISEASES OF THE HEART.

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### LECTURE I.

ON THE DIAGNOSIS OF CARDIAC DISEASE GENERALLY, WITH  
REFERENCE TO THE SYMPTOMS AND THE PHYSICAL SIGNS.

GENTLEMEN,—In gathering together the prominent facts in relation to the circulatory system in any case, few things are more striking than the apparent irrelevance of the symptoms in many of those patients who are otherwise ascertained to be suffering from serious cardiac disease, if we perhaps except the equally remarkable fact, that cardiac symptoms are frequently complained of when no cardiac disease exists. Thus, should a patient come to you complaining of palpitation, irregular action, or of his heart generally, you may at once assure him, without much fear of being wrong, that his heart is all right, that he is only weak, nervous, and probably dyspeptic. The exceptions to this rule occur in those past middle life and in old hospital patients, who, from having been so often lectured over, are almost as well acquainted with their own special lesion as an average practitioner.

Of course, if pain be a predominant symptom, that is usually referred to the region of the heart, but not always to the organ itself; and we continually meet with cases in which serious

valvular lesion has existed for many years altogether unnoticed by the patient, even although he may have been leading an active and laborious life. Such a lesion is said to be mute; that is, it has been perfectly compensated, and it never asserts itself as a disease until that compensation has been ruptured by accident, or by the gradual advance of those organic changes which are inseparable from it. In the one case the ruptured compensation is reparable, and comparative health may be restored; in the other, it is irreparable, though judicious treatment may prolong life and postpone the inevitable end.

A truly cardiac patient—one suffering from actual disease of the heart—as a rule comes to you complaining, not of that organ, but of one or other of the secondary results of his lesion. He complains of breathlessness or of dropsy, either or both of which may result from that lesion if it be insufficiently compensated, or if the compensation be ruptured.

If the patient complain of shortness of breath, as is often the case, you will find that this cardiac breathlessness presents certain distinctive features wherein it differs from pulmonary breathlessness, the most striking of these being the perfect tranquillity of the breathing while the patient is at rest, at the same time that any exertion at once produces so anxious a desire for more air as can be expressed by no fitter term than the *air-hunger* of the Germans. The amount of lesion is not to be measured by this breathlessness, but its seriousness, as dependent upon the degree in which the compensation is ruptured, may certainly be so. The patient may only puff considerably in going up a hill or ascending a stair, or his shortness of breath may be so great as speedily to compel him to call a halt on attempting either of these feats; or it may be so extreme as to prove distressing on making such perfectly trifling exertions as merely sitting up or turning in bed. At the same time there is no true dyspnoea, or difficult breathing properly so called; there is no obstruction either to inspiration or expiration; there may even be no curtailment of the air-space in the lungs from any cause whatever; the breathing while

the patient is at rest is perfectly quiet and natural : yet such is the difficulty—from cardiac causes—of getting the blood aerated, that the slightest exertion produces such a gasping inquietude as is extremely characteristic. This is one form of cardiac asthma, as it is termed ; now and then we have another in which the breathlessness, though not dependent upon exertion, is yet equally independent of pulmonary lesion. In this case the patient wakes gasping and alarmed from his first sleep ; he has palpitation, occasionally pain (angina), almost always irregular action of the heart, which is always feeble ; now and then the patient is sick, and sometimes vomits a mouthful or two. This form of cardiac asthma is mostly senile in character, and associated with muscular degeneration rather than with valvular lesion. It frequently arises from some slight gastric derangement, which reflexly affects the enfeebled heart in an injurious manner ; and it is often the beginning of the end to those affected—the first intimation that the “pitcher is broken at the fountain,” and that death has already seized the very citadel of life.

Such patients, however, never come to you—you are always sent for to see them ; and I have only mentioned this affection now to illustrate the fact, that exertion is not always necessary to produce cardiac breathlessness, and that even in this case the panting is characteristic, while the absence of pulmonary lesion marks its cardiac origin.

To produce so-called cardiac breathlessness, however, it is not necessary to have actual cardiac disease. Breathlessness depends upon imperfect aeration of the blood, and in the absence of pulmonary lesion may depend upon lesion of the heart or of the blood itself. Even though a patient, then, presents all the characteristic symptoms of cardiac asthma, we must not therefore set him down as certainly labouring under cardiac disease : he may be only anæmic. But inasmuch as anæmia and cardiac disease frequently coexist, the assured presence of the former, evinced by the bloodless condition of the lips, gums, &c., does not exclude the latter. The presence of a

breathlessness having the characteristic symptoms described, makes us certain that we have to do with a hæmic or a cardiac lesion; which it is, we must determine by further inquiries.

Dropsy depending upon cardiac disease always\* begins across the instep, and gradually fills up the lower extremities, the face and upper parts of the body remaining free. But inasmuch as the œdema of simple debility commences in the same position and pursues the same course, such a dropsy cannot be accepted as certainly indicative of the existence of cardiac disease, but must only be received as a hint that possibly the heart may be affected.

When a patient, then, who breathes easily when at rest complains of breathlessness on exertion, or of swelling of his feet, with or even without marked breathlessness, we suspect the heart to be the organ at fault, and we proceed to confirm or to set aside that suspicion by further investigation.

In further examining the condition of our patient, we first feel both radial pulses simultaneously, noting whether the arteries are firmer or more tortuous than usual (atheroma). If there be a marked difference between the two radial arteries, we feel both brachials simultaneously; if these be equal, the difference between the two radials is due to irregular distribution. If the brachials differ, in all probability there is some abnormal physical cause to account for it—possibly an aneurism, the mode of detecting which we shall afterwards describe. Should the radial pulses be equal and regular, but small and feeble, we elevate the wrist to a level with the head, if the patient be standing or sitting; if lying, we elevate the arm to its full length perpendicularly to the body. Should the pulse then become extinguished, or nearly so, the patient is anæmic, and possibly anæmia is his sole disease; but we must never under any circumstances rely upon one symptom, however apparently trustworthy, but merely note it as an aid and a guide in our further investigation. Should the pulse, after

\* "Almost invariably," says Walshe, "any exception being excessively rare."  
—"Diseases of the Heart," 3d edition, London, 1862, p. 302.

elevation of the arm, remain still small and feeble, but distinct, the cardiac disease, if present, is mitral. Irregularity of the pulse confirms this suspicion; extreme irregularity points to the probability of the affection of the mitral valve being constriction rather than simple insufficiency. Should the small, feeble, and possibly irregular pulse remain not only distinct after the elevation of the arm, but become more so, the systolic impulse being followed by such a sudden and complete collapse as to render the impulse apparently more marked, then we have to do with a double lesion, a mitral and also an aortic regurgitation. This form of pulse is, however, not always well marked, in many cases is not easy of detection, and is therefore not to be relied upon, unless the collapse is distinct. In simple aortic regurgitation, however, the peculiar sensation conveyed to the finger, and well known by the terms *water hammer* or *Corrigan's pulse*,\* is usually well marked, and frequently so greatly increased by elevation of the arm as to become almost painful, and wholly unmistakable.

We see, then, that while certain general symptoms indicate with greater or less probability the existence of cardiac disease, the examination of the pulse alone may not only confer more or less certainty on the suspicions thus aroused, but may even enable us in some degree to predicate the nature of the lesion.

We next examine the state of the patient by the INSPECTION of his thorax and neck, noting first whether there is any undue pulsation in the carotid arteries, or in the tracheal fossa; whether the veins are enlarged, and whether they pulsate or not. And to determine the latter fact in anæmic patients is sometimes a matter of some nicety, especially if they be young, as the veins are then small, and apt to be hidden by the subcutaneous fat, which is always more abundant than in older patients. When the individual is lying flat, a mere flicker at

\* *Vide* "Edinburgh Medical and Surgical Journal," vol. xxxvii., for April 1832, pp. 227, 229, where the phenomena referred to are described for the first time by the late Sir Dominic Corrigan.



the root of the jugular vein is of no importance, as it is found in most healthy people. A simple undulation in the jugular is a sign of considerable congestion of the right auricle, with propagation through it of the systolic impulse of the ventricle, the valves at the root of the vein remaining intact. But when we have distinct systolic pulsation propagated into the jugular veins, then we know that we have to do with dilatation of the right ventricle of some standing, which has rendered incompetent not only the tricuspid valve but also the venous valves at the root of the neck, and which must therefore have interfered injuriously with the systemic circulation. This venous pulsation is usually most evident in the right jugular vein, and is readily seen on pressing up the blood in the vein to the middle of the neck with the finger, the vein filling from below in a succession of waves synchronous with the cardiac pulsations.

Visible *venous* pulsation is therefore invariably a sign of considerable dilatation, with or without hypertrophy of the right ventricle, and its distinctness may be accepted as a measure of the persistence and degree of that dilatation; but visible *arterial* pulsation is occasionally found, especially in the peripheral vessels, in the normal condition, and is still more marked when these vessels are atheromatous and tortuous, as they frequently are in advanced age. But if we confine our inspection simply to the carotids, the tracheal fossa, and the brachials, and if we find well-marked, excessive, and symmetrical pulsation there, in the erect or semi-erect position, this will invariably be found associated with regurgitation through the aortic valves, and with coexistent dilatation and considerable hypertrophy of the left side.

Inspection of the chest in its normal condition reveals for the most part a perfectly symmetrical state of the thoracic walls, on both sides of the sternum. These walls gently rise and fall rhythmically with the in- and expiration, the only thing breaking the monotony of this gentle undulation being the tap of the cardiac apex—averaging four taps to each respiratory



wave—which is visible between the fifth and sixth ribs, about two inches from the left edge of the sternum.\* Any deviation from these appearances is abnormal, though possibly not of much import. Thus, in many cases, the precordial region is more prominent than the similar region to the right of the sternum. Frequently this is of no consequence, yet it may be associated with enlargement of the heart, or with pericardiac effusion if the intercostal spaces be effaced. Of itself, it is a sign of little importance; and in ascertaining its presence, we must be careful not to be misled by any rachitic bulgings of the ribs, or even by the more than usually distinct pulsations of the heart in children or in meagre individuals, which may apparently simulate a bulging. This latter simulation is readily corrected by more careful inspection, supplemented by palpation and measurement. In the rachitic chest the spinal column is usually found to be curved, or, at all events, the costal arch is deformed and depressed posteriorly when it bulges in front. Bulgings dependent upon arterial aneurisms invariably commence above the fourth rib, and appear as mere local tumours. Depression of the precordial region is much more rare than its elevation, and is the result of previous pericarditis, and the indication of adhesion of the visceral and parietal portions of the pericardium. We must also distinguish between a permanent and general depression of the cardiac region and those rhythmical depressions of the intercostal spaces which occur over the apex, or even over a more extended portion of the heart's surface, as the result of adhesions of the pericardium, not only to the heart, but also to the pleura, and through that to the walls of the chest, but which are sometimes, especially in thin-walled chests, simply the result of atmospheric pressure depressing the intercostal spaces at the moment of the cardiac contraction, where no adhesions exist; this form of rhythmical depression being invariably associated with some degree of enlargement—not always hypertrophy—of the heart and con-

\* Those who have worked much with female patients will appreciate the discarding of the nipple as a fixed point.

sequent displacement of the lung. Alterations in the position and extent of the apex beat are also readily appreciable by the eye. These must be noted accordingly, to be afterwards more fully investigated by palpation. Pulsation is also frequently seen in the epigastric region. Sometimes this is associated with absence of the apex beat from its usual position, and is to be regarded as one form of its displacement—a displacement which may be brought about in various ways, the commonest of these being dilatation of the right ventricle, by which the left ventricle is pushed backwards, the right one communicating its impulse to the lower part of the sternum and to the liver, which is then seen to pulsate in the *scrobiculus cordis*. It may be doubted if such pulsations are ever visible in a perfectly normal condition of the heart and neighbouring organs. Assuredly, they are often seen where no actual cardiac disease exists; simple dilatation of the right ventricle is invariably more or less present when pulmonary congestion exists, even from such simple and temporary causes as strenuous exertion or bronchial catarrh; and whenever dilatation of the right side exists to any considerable extent, epigastric pulsation may be seen. It is rendered more perceptible by any cause which may effectively conduce to the transmission of such impulse to the abdominal walls. Thus it is sometimes favoured by the existence of effusion in the pericardium, but especially by the occurrence of enlargement of the liver, which is so usual a concomitant of dilatation of the right ventricle. Not infrequently the systolic impulse of the venous regurgitation is so great as to induce expansile pulsation of the whole liver, a pulsation which is then visible, not merely in the *scrobiculus cordis*, but throughout the whole right hypochondriac region;\* an extent of hepatic pulsation which, apart from its expansile

\* This form of hepatic pulsation was first described by Allan Burns, in his "Observations on some of the most Frequent and Important Diseases of the Heart," Edin. 1833. At p. 265, he quotes a case from Senac, in which direct pulsation was communicated to the epigastrium by a vena cava inferior, the size of a man's arm. At p. 266, he also mentions a case in which he states that epigastric pulsation was produced by repercussion from solidified lungs.

character, is not, however, distinctive of venous regurgitation, as it is occasionally seen as the result of the impulse communicated by a large aneurism lying immediately above the liver.\* There are other pulsations in the epigastric region not depending upon cardiac impulse, such as the ordinary beating of the abdominal aorta, which occasionally becomes visible through the emaciation of those previously of full habit, or which may be transmitted to the abdominal surface by some overlying tumour. Now and then this pulsation is actually aneurismal in character : much more frequently it is simply neurotic, and yet limited to the abdominal aorta. Under some of these circumstances we can readily understand that the pulsation extends downwards along the course of the aorta ; under others, as when propagated upwards by a solid tumour, it is obvious that it may not only be limited in its longitudinal propagation, but may even extend transversely. Inspection directs the attention to these pulsations, indicating to an experienced eye their probable nature, which can only be accurately determined by other means of exploration. In rare instances, a pulsatory movement is also communicated to the epigastrium through the movement of the heart's apex during the ventricular systole, pulling upwards an adherent pericardium, diaphragm, and liver.† This movement is, of course, exactly the reverse of that in ordinary epigastric

\* Extract of a letter from a late physician labouring under aneurism of the thoracic aorta projecting through the sternum :—" I am satisfied that your view of the origin of the liver pulsations from the aneurism mainly is the correct one, however discouraging to myself that may be. But I have this to say, on the more cheerful side, that if an *enlarged* liver has had nothing to do with them (as Dr — says), and which by decreasing in bulk has caused them to be less felt, the very great decrease in them which has occurred since I began the iodide, is very favourable to the conclusion, that under its action the sac must have contracted considerably. Early in February, before I had reason to suspect any increase of the liver, the pulsations had the effect of expanding the opposite sides of the hypochondria at each beat, an effect which is not now perceptible, though I am supposing the liver to be larger, in which, however, I may be mistaken." Dated 3d May 1870.

† A remarkable instance of this will be found detailed in a note on p. 214 of " Copland's Dictionary of Practical Medicine," vol. ii.

pulsation. As this extensive adhesion is, as a rule, the result of a severe and extended inflammation, affecting the whole surface of the heart, we can readily understand how, in such circumstances, a universal undulatory movement may be perceived, in which, when the heart's action is at all rapid (over 90 beats per minute), it may be difficult to say what parts of the motion are systolic and what are diastolic. In these circumstances, the variations in time between the movements of any two parts are readily rendered visible by attaching to each, by means of a pellet of beeswax, a bristle carrying a small paper flag; and when the pulse is over 90, this is the only way in which such differences can be ascertained with any certainty; and it is a means of attaining accuracy in diagnosis often of much importance, especially when we have pulsations visible above the fourth rib, which may be either aneurisms of the aorta or pulsations of the auricle. A comparison of the movements of a flag on the doubtful point with one over the apex, will settle the matter at once, as the beat of the auricles always precedes that of the ventricle, while, however near the heart an aortic aneurism may be, its pulsation can never precede that of the ventricles, but must either be synchronous or succeed it by an interval which is more or less appreciable. The determination of this fact, we shall afterwards see, may be a matter of some importance in the diagnosis of cardiac lesions.

PALPATION confirms the information obtained by inspection, and adds somewhat to it. On placing the hand over the cardiac area, in many people with thin chest walls, we can distinctly perceive the alternate movements of auricular and ventricular systole, with each corresponding diastole; and, of course, we can also readily appreciate any pathological change in these movements. Great increase in their force indicates hypertrophy; but a diminution of that force is by no means to be regarded as a certain indication of atrophy, dilatation without hypertrophy, or even of cardiac debility from any cause,—though it may be a sign of one or other of these pheno-

mena, as well as of great pericardiac effusion, pulmonary emphysema, or mere increased muscularity of the thoracic walls. In many of these cases, especially in pericardiac effusion, but more or less in all, the cardiac shock may be rendered perceptible to the hand, by causing the patient to sit up and lean well forwards. In this case, the readiness with which it can be appreciated, and the position in which it is felt, must be carefully noted. Forcible pulsation above the fourth rib, and within the cardiac area, may be aneurismal, but if to the left of the sternum it more commonly depends upon dilatation and hypertrophy of the appendix of the left auricle. Forcible pulsation chiefly to the left of the cardiac area below the fourth rib, with depression of the apex beat, indicates dilatation and hypertrophy of the left ventricle; while pulsation beneath the lower part of the sternum, with disappearance of the apex beat, reveals dilatation with or without hypertrophy of the right ventricle, the extent of dilatation being to some extent measurable by the amount of epigastric pulsation, while the degree of hypertrophy is denoted by the force of the pulsation. When the whole heart is hypertrophied and dilated, a more or less violent shock may be felt over the whole of the cardiac area; and when this hypertrophy is great, the shock may be double—the first forcible and systolic, the second less forcible and diastolic,—the result of the rebound of the enlarged heart from the posterior thoracic walls.

Displacement of the apex beat may be produced in the normal state by inclining the patient to the left side, when it may pass to the left a little farther even than a point equidistant between its normal position and the axillary line; and by inclining the patient to the right, the apex beat becomes faint or disappears, the right side of the heart being then felt to give a distinct pulsation in the epigastrium. In pericardiac effusion, the apex beat—or what seems to be so—may be displaced upwards as far as the fourth interspace, and slightly outwards to the left. In pleural effusion on the left side, it may be turned quite

round the other way into a similar position on the right side, and pleuritic effusion on the right side may displace it towards the left. In hypertrophy, with dilatation of the left ventricle, the apex beat passes downwards and to the left. In a similar condition of the right ventricle, it may entirely disappear from its normal position, a diffuse impulse at the lower end of the sternum revealing the cause.

When the left lung is retracted from the base of the heart from any cause, the pulsation of the pulmonary artery between the second and third ribs, close to the sternum, may be distinctly felt, and even the click of the semilunar valves perceived, because sound consists of vibrations sufficiently rapid to be appreciated by the ear, and may be both felt and seen if the propagating medium be suitable. Hence the palpating hand can readily perceive the friction resulting from the rubbing together of the two layers of the pericardium roughened by lymph, and may even recognise the vibration of valvular murmurs. When rough, these vibrations have received the name of the purring thrill—*fremissement cataire*—because the sensation felt resembles that perceived on placing the hand on the body of a cat in the act of purring. Abnormal pulsations along the course of the thoracic aorta are usually confined to the ascending and transverse portions, and may be the result of simple displacement of the vessel, as occasionally happens in rickety chests, or they may be caused by aneurismal dilatations, and must be sought for in the tracheal fossa, as well as between the ribs along the course of the aorta, especially during expiration, when such pulsations, if faint, are most readily felt.

From all this we see that a very great amount of information may be obtained from palpation, a great part of the diagnosis frequently depending upon the information thus received. The points to be specially noted are—1<sup>st</sup>, The position of any perceptible pulsation; 2<sup>d</sup>, Its extent and force; 3<sup>d</sup>, Its rhythm, whether systolic, diastolic, intermittent, or irregular; and 4<sup>th</sup>, Whether any vibratile sensations are

perceptible over the seat of pulsation, and if so, what is their apparent rhythm.

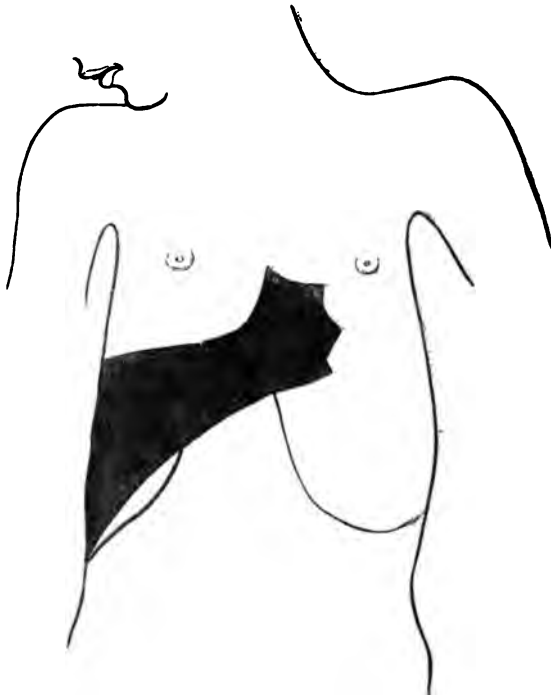
Having thus registered all the information to be obtained from the inspection and palpation of our patient, we now proceed to PERCUSS him, and from this we procure information of a totally different character from any hitherto obtained. Up to this time we have been dealing with phenomena which can be seen and felt, but by percussion we can map out the entire cardiac organ, or at least we can limit with very considerable accuracy the extent of the cardiac dulness even in those parts where movement is unseen and unfelt; and, as we shall by-and-by see, we thus possess ourselves of a most important link in the chain of facts which connects those visible and tangible phenomena with that hidden cause, the condition of which it is our object to elicit. Percussion of the heart is usually reckoned rather a difficult and unsatisfactory procedure; in reality, if we set about it in a right way, and correctly interpret its indications, it is not only one of the easiest but also one of the most instructive problems to be found in the varied applications of this method of diagnosis. All that we can learn from percussion is simply the size, form, and position of the precordial dulness, but we can learn these accurately, and, as already said, these are facts which may materially aid us in our inquiry.

Percussion, in medical parlance, is the art of ascertaining the condition of the internal organs of the body, as to resonance, by percussing or tapping upon the surface of the body just exterior to the organ whose state we wish to examine. It is obvious that this condition may be modified, first, by the amount of air contained beneath the part percussed—the size of the resonant cavity; and, second, by the physical condition as to tension and structure of the parts on which we operate; and, in the living body, all of these circumstances may be variously modified. The heart itself, and the large blood-vessels with which it is immediately connected, contain, in the normal state, no air, and are never sufficiently tense to be

themselves thrown into sonorous vibrations by percussion ; when struck, therefore, mediately or immediately, they give forth no sound at all, or are simply said to be dull on percussion. The heart and large vessels, however, in their natural position, are surrounded on three sides by the lungs, which are normally filled with air, and are therefore resonant ; while, on the fourth side, the heart rests upon the liver, like itself, a dull, non-resonant body, from which we can, however, separate it more or less perfectly by measures presently to be described.

On percussing over the left side of the thorax, the veriest

Fig. 1.



tyro can at once distinguish a more or less triangular area of complete dullness (fig. 1\*), which varies in size and shape in

\* This figure is a diagrammatic representation of the cardiac and hepatic dullness of fig. 4—organs healthy—of “Sibson’s Illustrations of the Morbid Anatomy of the Organs of the Chest,” published in the twelfth volume of the



each individual, and is an exact measure of that portion of the heart in contact with the thoracic walls. The magnitude of this area of superficial dulness, as it is termed, is often taken as an indication of the actual size of the heart itself, but it obviously only betokens the comparative degree to which the heart is uncovered by the lung, and as that may depend either upon increased size of the heart itself or diminished expansion of the lungs, the information conveyed as to the size of the heart is of no positive value whatever. To ascertain this accurately it is evident that we must have recourse to mapping out the entire area of cardiac dulness, both superficial and deep, to use for the nonce those anatomical terms which are so constantly applied to acoustic phenomena, in spite of Auenbrugger's\* protest that they ought to be explained and described solely by reference to the physical condition of the parts implicated, and not by any reference to their anatomical position, that being a matter not immediately apparent from the sounds elicited, though it may be deduced from them by a process of ratiocination.

The heart and large vessels occupy the centre of the chest, extending from the upper border of the third rib to the lower end of the sternum, and almost entirely fill up the space between the sternum and the vertebræ. In this position the ventricles encroach more upon the left lung, and the auricles upon the right one, the whole of the right cavities lying anterior and slightly to the right of the left ones ; the axis of the right ventricle in relation to the pulmonary artery being almost vertical, the ventricle being broadest at the part most distant from the artery ; while the axis of the left ventricle is almost horizontal in relation to the aorta, the ventricle being

"Transactions of the Provincial Medical and Surgical Association." It has been selected because the area of cardiac dulness approaches more nearly in configuration the ordinary conventional idea of such dulness than that of the other figures. How much this may vary within the limits of health may be seen by a reference to the other figures taken from bodies with healthy organs.

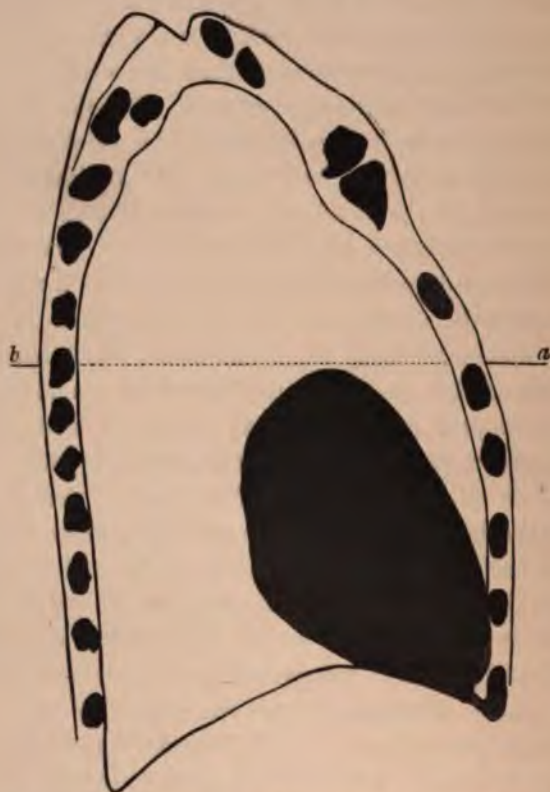
\*"Inventum Novum," §§ 17, 18, scholia. Auenbrugger was the first to apply the art of percussion to the discrimination of diseases of the chest. The earliest edition of his work was published at Vienna in 1761.

narrowest at the part most distant from the artery ; in consequence of the peculiar formation of each ventricle, and of the anterior position of the right one, the pulmonary artery arises in front and to the left of the aorta. From the formation and position of the heart, it is obvious that, though we can and may percuss out the whole of the cardiac dulness, this is quite unnecessary ; it is only of importance to ascertain its greatest extent of dulness vertically and transversely. Increase of the vertical dulness rarely indicates any alteration in the size of the heart itself, but is usually either due to effusion into the pericardium, or to hepatic enlargement, readily ascertained by an extension of this exploratory method to the liver itself ; the former dulness extending above the third rib, the latter, as a rule, below the sixth ; while a simple change of position of the heart, which may arise from various causes, is indicated by a transference of the normal dulness upwards or downwards, without any change in its extent. The apex beat, except in certain abnormal conditions, is, from the formation of the heart, the part which extends farthest to the left, and being, as a rule, perceptible to the touch, only requires to be percussed out in those exceptional circumstances where the true apex beats beneath a rib, and not in an interspace. The right auricle is, of course, that part of the heart which extends farthest to the right, and being extremely dilatable, and readily influenced by any obstacle to the onward flow of the blood, the transverse dulness at the level of the fourth rib comes to be an important indication of some obstacle to that onward flow, and therefore of enlargement of the heart chiefly in its auricular region. These, therefore, are the main points in regard to which we look for important information from the percussion of the cardiac dulness. Increase of dulness above the third rib indicates, as a rule, pericardiac effusion. Increase of the transverse dulness at the level of the fourth rib indicates obstruction to the circulation. If the apex beat be displaced to the left and downwards, the obstruction is probably aortic, and has primarily influenced the left ventricle ; if the apex

beat be not displaced downwards, the obstruction is either mitral or pulmonary in its origin. In percussing the cardiac dulness, we must always remember that, so far as the three sides surrounded by the lungs are concerned, the passage of the percussion note from clear to dull is not abrupt, but transitional, because the heart upon all these three sides is covered by a gradually decreasing layer of lung. Practically, we find, in percussing the cardiac region, that the percussion note from above downwards ranges from clear and full just above the upper border of the third rib to perfectly dull somewhere below the lower border of the fourth rib, the note becoming gradually less full, but still perfectly clear, until it reaches the limit of superficial dulness. As this diminution of fulness depends upon a lessening of the air-space, Skoda has applied to it the term *leer* or empty, the percussion sound becoming gradually emptier by the filling up of the resonant air-space from below, while it may also become gradually duller—more muffled—by filling the air-space from above,—that is, by increasing the density of the medium percussed, whose vibrations originate the sound, a perfectly empty sound and a perfectly dull one being, of course, synonymous; the empty sound, however, remaining clear to the last, becoming gradually emptier until perfect emptiness and perfect dulness coincide; while a muffled or originally dull sound may be full at first, but becomes gradually emptier till perfect dulness and perfect emptiness coincide. The first form of dulness, the emptying of the sound, depends upon a gradual diminution of the air-space; the second form, the muffling or dulling of the sound, depends upon the gradually decreasing capacity of the percussed medium for undergoing sonorous vibrations. A percussion sound becomes, therefore, gradually emptier, less full, the duller or more muffled it becomes; but it by no means necessarily grows duller by becoming emptier, because a sound may be very empty, and yet perfectly clear. It is obvious that, in this sense, the terms full and empty are equivalent to greater or less intensity of sound, the quality of

intensity depending upon the quantity of sound reaching the ear. A glance at the accompanying diagram (fig. 2\*) will

Fig. 2.



show that, in the normal condition, we have above the third rib (along the line *a b*) a larger air-space—a larger resonance

\* This figure is a diagrammatic representation of fig. 2. tab. 5 of Fasciculus 2 A of Pirogoff's "*Anatomia Topographica Sectionibus per Corpus Humanum congelatum triplici directione ductus illustrata.*"—Atlas, Petrop., 1859. The section passes at the upper part through the left sterno-clavicular articulation, at three Paris lines from internal margin of the head of the left clavicle. The lowest rib anteriorly is the seventh, cut through ten lines from the sternum; posteriorly, it is the twelfth rib, cut through nine lines from its vertebral extremity. It will be observed that, in this figure, the lung extends lower than ordinarily, and that perfect dulness would not commence till the lower edge of the fifth rib, instead of the fourth rib as usual.



box, and therefore a fuller sound producible on percussion—than below the third rib, and that the air-space gradually lessens, and, consequently, the percussion note gradually becomes less full, as we approach the lower border of the fourth rib, continuing to be clear up to the very last. In most works upon the subject, we are told to percuss more forcibly below the third rib, in order to elicit what is termed the *deep dulness*; but the idea of deep dulness is a composite conception with which acoustics have only a relative connection; all we require to ascertain is simply the size of the resonance box—the greater or less fulness of the percussion sound; and in the average normal condition of the chest-wall and the contained organs, the slightest tap is sufficient to elicit the difference between a full and a less full or emptier percussion sound. When the chest-walls are denser or less vibratile, and the percussion sound consequently more muffled, a more forcible percussion may be required; but this refers equally to the full and to the less full sound; is the same, therefore, as regards percussion above the third rib and below it.

Acoustic phenomena are generally spoken of in relation to their intensity, pitch, and clang or *timbre*. By many observers the phenomena just referred to *intensity*, and spoken of as full or empty sounds, have been attributed to pitch, and regarded as low or high sounds; and indeed an emptying of the sound is necessarily accompanied by a heightening of its pitch. Because the length of a column of air which most perfectly resounds to the vibrations of a tuning fork is exactly equal to one-fourth of the length of the sound wave produced by the fork, and this wave increases in rapidity as it diminishes its length, increase in rapidity being equivalent to heightening of pitch, as we learn from experiments on the syren. Hence the shallower any air-space, the higher the note with which it most perfectly resonates, a percussion note becoming emptier becomes therefore also higher in pitch. Not that there is any alteration in the general rapidity of the vibrations of the membrane percussed because the resonance box over which it is

stretched is shallow, but because the air-space beneath the part percussed selects those flutters with which it resonates, and raises them only to the dignity of an audible tone. Pitch is that quality of sound most readily recognised by the average ear, it is also a strictly acoustic term which dulness is not, it is therefore sufficient for the student to note the alterations in pitch obtained on percussing the chest-wall, assured that whenever the pitch obtained on percussing the chest-wall is found to rise, the air-space is diminished proportionately. A knowledge of anatomy will enable the student to know whether his phonographic map of the chest-wall is normal, or whether he will require to call pathology to his aid to explain the abnormal alteration. *Clang*, on the other hand, depends upon the mingling of the overtones or harmonics belonging to the vibrating body itself with its fundamental note, and therefore varies with the nature of the structure thrown into sonorous vibrations by percussion—varies, therefore, as we percuss the intercostal tissues; the ribs; or the sternum—varies even with the structure of the pleximeter or plessor employed. Nay, more, clang, mingled with resonance, has its influence in altering the percussion note in those cases where, from emaciation and the size of the pleximeter, the latter rests upon two ribs, leaving an air-space of varying size beneath. As clang, therefore, varies with structure, tension, and elasticity, it is advisable—to avoid its confusing influence in estimating the exact quality of a percussion note—in percussing from above downwards, to compare rib with rib, and interspace with interspace; and, for the same reason, in percussing transversely, we must carefully percuss along a rib or along an interspace, and avoid shifting indefinitely from one to the other. Both pleximeter and plessor must also be as much as possible free from clang themselves, and the former must be capable of close and accurate application to either rib or interspace; and in these respects no instruments are comparable to the forefinger of the left hand for a pleximeter, and the first two or three fingers of the right hand as a plessor,



the only objection to their use—and it only applies to class teaching—being the comparatively less intensity of the sound produced; but this difficulty is easily surmounted by the use of a Winterich hammer as a plessor, by means of which an adept can, without injury to the finger pleximeter, produce a percussion note of perfectly sufficient intensity. A less skilful party using such a hammer is apt not to tap smartly enough; he may hit hard enough—there is not much likelihood of failure in that respect; but he does not lift the hammer quickly enough, and he therefore muffles or stops the sonorous vibrations it is his object to excite. It occasionally happens that the rise in pitch due to the right auricle is not appreciable to the right of the sternum, and under these circumstances the clang of the sternum may be so sonorous as—in percussing transversely—apparently to do entirely away with all cardiac dulness; but, unless under very exceptional circumstances, we can remedy this matter by percussing from above downwards; and by eliciting the sternal note above the aorta, we can readily recognise the rise in pitch due to the heart lying beneath the sternum. We must also never forget that the arch of the aorta does not extend like a bow transversely across the sternum, as Piorry has figured it, but that, as anatomy teaches us, it is in close contact with the pulmonary artery and the heart at every point, so percussion can only map it out as a somewhat rounded projection at the base of the heart. We must also remember that, as a rule, the dulness of the innominate artery is scarcely perceptible, while that of the left carotid, and that of the left subclavian, is altogether imperceptible. For this the clang of the ribs lying over them is partly to blame; but it is, no doubt, chiefly due to what is called the inflection of sound, by which a sonorous wave embraces a non-resonant body on all sides, and may, so far, extinguish the dulness of a small one as to render it inappreciable. Hence marked dulness in these regions is usually significant of some considerable morbid alteration.

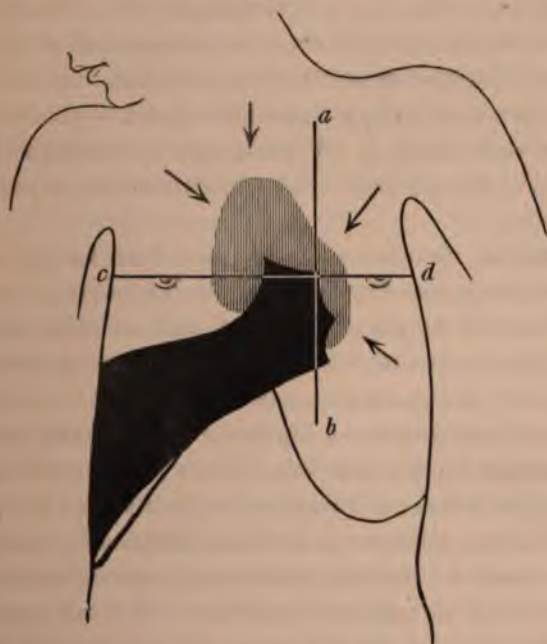
As the heart rarely rises above the level of the third rib, and if it does so, it rises, as a rule, equally on both sides, so it is obvious that Piorry's line of oblique dulness, from apex to base, is of no special practical value; and, from all we have said, it follows as a corollary that there are only two lines of percussion of any practical importance, viz., a vertical and a transverse one. The first of these must be thrown so far to the left as to be uninfluenced by the aorta and the pulmonary artery, and I have been in the habit of placing it at one inch to the left of the left edge of the sternum. When, percussing from above downwards from the lower edge of the left clavicle, we have in the normal condition first the clear, full sound of the lung, low in pitch and of considerable duration down to the upper border of the third rib; beneath that, the percussion note gradually rises in pitch and shortness in duration, becoming, as it is called, gradually emptier, but still clear down to the lower border of the fourth, or upper border of the fifth rib; and beneath that we have perfect dulness, till we reach the tympanitic clang of the stomach or intestine. Any deviation from this is abnormal, and its explanation must be sought by other methods of investigation. In like manner, the transverse dulness in the line of the fourth rib, which is that mainly affected by distention of the right auricle, must be carefully mapped out, percussing from without inwards on each side—that is, from the lung to the heart; because in that way alone we can appreciate the gradual rise in pitch, and also eliminate the clang of the sternum. The accompanying diagram (fig. 3\*) exhibits the percussion dulness of the normal cardiac area, with the lines of practical importance (vertical, *a* to *b*; transverse, *c* to *d*) specially indicated. To separate the lower part of the heart from the liver is by no means an easy matter. It is said that we can occasionally make out a line of clearness between the two. I have never observed any such line of clearness, and, as the heart invariably rests upon the diaphragm,

\* This diagram refers to the same subject as figure 1. Percussion is to be made in the direction of the arrows.



unless separated by fluid, I do not see how this line of clearness can be produced. Any change of pitch between the cardiac and hepatic dulness is inappreciable, except that produced by upward conduction of the tympanitic note from the stomach or intestines. It follows, then, that we can only separate the heart from the liver approximately, by ascertaining the position of the apex beat upon the left side, and the highest point of hepatic dulness upon the right side, and joining the two by a straight line. This line of separation can

Fig. 3.



never be absolutely accurate, but is usually sufficiently so for all practical purposes.

It is sometimes of importance to map out the aortic dulness, so as to ascertain if it be increased in any direction; and this is by no means a difficult task, if we set about it in a right way. As I have already said, it is impossible to separate the aortic dulness from that of the heart: the only part in which

this might be possible is occupied by the pulmonary artery, the dulness of which differs in no respect from that of the aorta. It is therefore absurd to map out the aortic dulness, as Piorry and his followers have done, as an independent arch crossing the chest above the area of cardiac dulness. The normal aortic percussion is indicated in the foregoing diagram (fig. 3), in which it will be seen to occur simply as a more or less rounded area of dulness rising out of and inseparable from that of the heart, and only known to be aortic from its position above the upper limit of cardiac dulness in the line *a b*. Increase of this dulness in any direction may depend upon morbid enlargement of the aorta; but in the diagnosis of these enlargements, percussion dulness is only one of many physical facts which require to be collated and reasoned upon in estimating the probabilities for and against the assumption of the existence of any such morbid condition.

Absence or diminution of the precordial dulness in the rarest of cases is caused by the presence of air in the pericardium, less seldom by atrophy of the heart, and infinitely more frequently by increase of the area of pulmonary resonance due to emphysematous expansion of the lung. Increase of the area of cardiac dulness is a much more common phenomenon. This may depend upon effusion into the pericardium, in which case the dulness is pyramidal with the base downwards and the cardiac pulsation is annulled, enfeebled, or displaced. Or the increased dulness may depend upon enlargement of the heart with or without hypertrophy; in these cases, the pyramidal shape of the dulness is less marked, but its broadest part is always above. When hypertrophy predominates, the cardiac shock is increased, when dilatation predominates, it may be almost annulled. But we must never forget that these conditions may be variously modified, and that our diagnosis, to be correct, must be based, not upon one or two facts, but upon all the phenomena which can be ascertained in regard to the physical condition of the heart.



The next and last method\* which we can employ in ascertaining the physical condition of the heart is AUSCULTATION, by which we understand the art of ascertaining the physical condition of the mechanism of the heart by the sounds produced during the passage of the blood through its several cavities.

If we place our ear over the cardiac area during the progress of the circulation, we become conscious that it is accompanied by sounds alternated with silences. If we listen over the heart of an infant, we distinguish only a uniform ticking, in which the sounds are alike in intensity, and the silences similar in duration; but, as we gradually extend our experience by listening to the hearts of older individuals, we find that, as we approach adult life, these sounds, with their intervening silences, assume a peculiar rhythm, which the ear thus educated very readily appreciates; and we also discover, that while the silences and the sounds remain the same in every position, their rhythm varies according to the part of the cardiac area in which they are heard. Thus, when listening over the apex beat, we distinguish two pauses or intervals of silence,—a long pause and a much shorter one; immediately succeeding the long pause, we hear a dull, prolonged sound, followed by the short pause, and this succeeded by a much shorter and sharper sound, which is immediately followed by a renewal of the long pause, the accent in this situation being upon the prolonged or first sound following the long pause, making what is called a trochee in prosody “— ∪.” On the other hand, when we listen over the base of the heart, we find that, while the relation of the sounds to the silences remains the same, the accent in this situation falls upon the second sound instead of on the first, so that, following the long pause, we have, instead of a trochee, an iambus “∪ —.” We see thus, that though the cadence of these sounds alters with the position in which they are heard, they can, in the normal

\* For, of course, in the practical exploration of the heart, we exclude both the cardiograph and the sphygmograph, the use of which can never become general as a means of clinical examination by an ordinary practitioner.

adult, be readily enough recognised to be first and second by their relation to the periods of silence; but whenever the heart's action becomes rapid from fever, debility, or any other cause, the long pause is diminished, and the rhythm of the sounds approaches that of the infant, so that, when the pulse beats more than 90 times a minute, it is almost impossible to distinguish with accuracy what is first and what is second, and if for any reason it is desirable to do so, we must employ a double stethoscope, and by placing one end over the heart's apex, and the other over the base, a little practice and a little care will enable us to distinguish what is first from what is second, by a due attention to the slight difference in the character of the sounds, and by a careful appreciation of the position in which each sound is most distinctly heard. Apart from the alterations in rhythm, which are due to the rate of cardiac action, the heart sounds vary in distinctness in each individual; and experience has taught us, that in hearts which are thin-walled, the cavities of which are relatively large in proportion to the thickness of the walls, whatever that may be, the first sound is particularly loud, clear, and distinct; while in hypertrophied, thick-walled hearts, the same sound is always muffled and indistinct; the second sound not being affected by these causes. Of course we cannot securely base our diagnosis of the state of the cardiac walls upon the distinctness or indistinctness of the first sound; but this phenomenon supplements and confirms the information we obtain in other ways. The second sound also varies in distinctness and in intensity, but always from causes which are extracardiac.

Besides the alterations in rhythm depending upon alterations in the rate of cardiac action, and alterations in distinctness depending upon hypertrophy, dilatation, or some extracardiac cause, we find these sounds occasionally replaced by noises, *bruits*, or murmurs, as we term them, which are totally different from the sounds they displace; and which vary with their causes.

Physiologists teach us that the first sound coincides with



the contraction or systole of the ventricles, and is composed of several ingredients, of which the shock of the heart's apex on the walls of the chest, the bruit which accompanies muscular action, and the sound produced by the closure of the auriculo-ventricular valves, are the most important. But for all practical purposes the last is all-sufficient, as we find that whenever these valves are from any cause—such as dilatation of the ventricle—rendered incapable of closure, the valves themselves being healthy, the first sound is either partially or wholly replaced by a murmur which more or less completely obscures the muscular bruit, and, to a less extent, the shock. The second sound, on the other hand, as it immediately follows the ventricular systole, must coincide with the ventricular diastole, and therefore with the moment of time when the arterial semi-lunar valves are closed by the recoil upon them of the blood compressed by the arterial systole; and, accordingly, we find that when these valves are from any cause rendered incapable of closure, the second sound is more or less completely replaced by a murmur according to the degree of imperfection present.

The heart, however, is a double organ, with two auriculo-ventricular openings and two large arteries springing from it; and it is of importance to determine which side is affected. But it so happens that all the cardiac valves lie so close together, that a superficial area of half an inch square will include a portion of all the four sets of valves *in situ*, while an area of about one quarter of an inch will include a portion of all except the tricuspid.\* It is obvious, therefore, that it is impossible to differentiate the sounds or murmurs produced by one valve from those produced by another by merely listening with an ordinary stethoscope over the place of origin. By the rhythm alone we may readily distinguish which is first and which is second; but to differentiate a right first or second sound from a left first or second sound, we must take means to separate the one from the other; and this we do by taking advantage of the facts that sounds produced in one medium

\* Walshe, *op. cit.* p. 6.

lose in intensity in passing into another, and that sounds produced by any fluid in motion are invariably transmitted in the direction of the onward current.\* Hence we find that sounds produced in any one cavity of the heart are usually heard with most distinctness over that part of the thoracic wall at which the given cavity approaches the surface most closely. Thus, the only point at which the left ventricle directly impinges on the chest-wall is where the apex beat is felt, and that is precisely the spot where the first sound produced in the left heart by the closure of the mitral valve is most distinctly heard: a space of about one inch in diameter around the apex beat is therefore termed the mitral area. Nearly the whole of the right ventricle is uncovered by lung, and impinges directly on the lower part of the sternum; and at this part, especially along the left edge of the sternum, where it is joined by the cartilages of the fourth, fifth, and sixth ribs, the right first sound produced by the closure of the tricuspid valve is best to be heard, and the triangular space covering the position of the right ventricle is therefore termed the tricuspid area. In the normal condition, the two ventricles act simultaneously; and the two sounds differ so little from one another, that it is impossible to differentiate clearly the one from the other. When, however, from any cause, these sounds become irregular or replaced by murmurs, the differentiation by means of a reference to these areas of audition is readily

\* Sound is reflected, inflected, and refracted like light, and is readily conveyed to almost any distance in smooth tubes; the difficulty which it finds in passing from one medium to another, even though these should be only strata of the atmosphere of varying densities, is sufficiently indicated by the curious statement that battles have been lost for want of reinforcements which were actually waiting within what was thought to be earshot for the sound of the cannon to indicate the moment of advance.—*Vide* Tyndall "On Sound," London, 1869, p. 23, &c. A most remarkable instance of acoustic opacity of the atmosphere, associated with optic lucidity, is to be found at p. 234 of the "Philosophic Transactions" for 1874, vol. 164, pt. i., where it is recorded that Mr M'Kean and General Randolph were at a distance of one mile and a half, for two hours, spectators of the battle of Gain's Farm, in which 50,000 men, and 100 field-guns were engaged, without hearing a single sound, yet in a different direction the cannonading was distinctly audible 100 miles away.



enough made, though, of course, there are always plenty of confirmatory proofs.

On the other hand, there is, both in health and in disease, a marked difference between the aortic and pulmonic second sounds, and it is often of the greatest importance to differentiate one from the other, and clearly to recognise the distinctive characteristics of each. As already mentioned, the aorta and pulmonary artery originate very close to each other, the pulmonary valve lying about the middle of the third left cartilage, one-half being to the left and the other to the right of the left edge of the sternum, which exactly divides it in two. From its point of origin, the pulmonary artery rises to the lower edge of the second left cartilage, where it divides into its two great branches going to the right and left lungs respectively; this, therefore, is the place where it is nearest the surface of the chest, and the second interspace, or still better, the sternal end of the third cartilage, is the position in which the pulmonary second sound is best heard. On the other hand, the aorta rises a little below, behind, and to the right of the pulmonary artery, its valve corresponding to the lower edge of the third left cartilage, behind, and to the right of the pulmonary valve, and it passes upwards, forwards, and to the right, till it reaches the upper border of the second right cartilage, when it passes obliquely backwards and to the left, forming what is termed the arch of the aorta. At the second right cartilage therefore the aorta is nearest the surface of the chest, and the arterial walls and the blood-current coincide in readily conveying in this direction the resonant vibrations which result from the closure of the aortic valve; in this position, therefore, the aortic second sound is most readily differentiated from the pulmonary one. In the normal condition the aortic second is heard to be louder and more distinct than the pulmonic one; both vessels are, however, normally covered with lung, and as, from various causes, congenital or morbid, one or other may become uncovered, and thus brought nearer the surface, its corresponding sound may then be heard

more distinctly than is normal, and the ordinary condition may be either intensified or reversed; but this does not happen without the occurrence of other phenomena, which enable us to detect and explain the source of the abnormality, as we shall by-and-by have occasion to see. But besides this greater distinctness of the one sound more than the other, depending upon mere alteration in the anatomical relation of the parts, we have occasionally an alteration in the character of one or other of these sounds depending upon intrinsic causes. To this peculiar alteration of the second sound a variety of terms have been applied: it has been called booming, ringing, clanging, pumping, cavernous, and accentuated; and, perhaps, accentuated or booming are the most unobjectionable expressions we can employ to define this particular change in the second sound, in which the element of tension seems united with an increase of distinctness—a combination which, however, must be heard to be properly understood. This accentuation of the second sound may be heard either at the second right or at the third left costal cartilage; it may, therefore, be either of aortic or pulmonic origin. Its occurrence in the pulmonary artery was long ago pointed out by Skoda as an important aid in the diagnosis of mitral disease; but it is not only in mitral disease that the presence of an accentuated pulmonary second is of important significance, for this accentuation is constantly present in every form of cardiac disease involving obstruction to the onward flow of the blood, and is the most persistent of all the acoustic phenomena indicative of cardiac disease, being occasionally the only thing markedly abnormal to be detected. In the absence, therefore, of any pulmonary disease capable of producing congestion, persistent accentuation of the pulmonary second sound is to be regarded as invariably indicative of cardiac valvular lesion. The mode in which this accentuation is produced is very simple. The comparative trifling difference between the arterial and venous blood pressures in the pulmonary circuit necessitates of course a very slight resistance in the pulmonary capillaries, in



order that there may be through them, in any given time, a blood-flow equal to that which passes through the capillaries of the systemic circulation where very different conditions prevail. From this absence of capillary resistance, as well as from the non-existence of vaso-motor nerves within the lungs, it follows that mere mechanical influences play a much more important part in the pulmonary circulation than they do in that of the system generally. The result of this state of matters is that no obstruction to the bloodflow, either through arteries or veins, can occur anywhere in the pulmonary circuit without raising the blood pressure within the pulmonary artery. But the blood pressure cannot rise within the pulmonary artery, without the pulmonary valve being closed with a greater force than usual; I suppose it is impossible to detect the lesser degrees of this, but so soon as the pulmonary second equals or exceeds the aortic second in intensity of sound, then we can have no doubt about it. In the systemic circulation the conditions are very different, but it is unnecessary to say more upon this, it is enough for the present to point out that as the aortic second is always louder and more distinct than the pulmonary second in the normal state of the circulation, it is even more difficult to detect any slight increase in its intensity. We have nothing with which to compare it, and what one man may regard as accentuated, another may think only normal. It is only when we have some distinct quality superadded, such as that which is very fairly expressed by the word *booming*, that we can speak with perfect confidence, and my own experience is that whenever this is heard we have always to do with some degree of dilatation of the ascending aorta. Sometimes we can detect this by other means, often we cannot, but if in a quiescent state of the circulation we can detect distinct accentuation of the aortic second, we may be quite certain that we have to do with some degree of aortic dilatation. Accentuation of the pulmonary second, unaccompanied by disease of the lungs, is therefore an invariable sign of some cardiac lesion; while accentuation

of the aortic second is as invariable a sign of aortic dilatation, when these accentuated sounds are heard in their normal positions,—the third right and the second left costal cartilages.

Outside of the normal area of cardiac dulness, as depicted in fig. 3 (p. 23), we occasionally hear the normal cardiac sounds more distinctly than is usual, and this may depend either upon increased resonance from condensation of that part of the lung over which they are heard, or upon an aneurismal bulging of the aorta at that part. In the latter case, we may either hear the normal cardiac sounds louder than usual in the given position, or if the cardiac sounds be abnormal, these abnormal sounds may be heard more distinctly than they ought to be; or the second sound alone may be heard accentuated in this abnormal position, or this abnormally placed accentuated second sound may be preceded by a localised systolic bruit, or it may be more rarely replaced by a localised diastolic bruit. But these sounds, when heard outside of the normal area of cardiac dulness, indicate disease of the lungs or of the aorta, if they be identical with those heard within the cardiac area; if they vary from them, then they indicate disease of the aorta in one or other of its various forms, and in any case have only to be noted for further examination. In the normal condition of the heart, the sounds and silences succeed one another in the manner described,—each ventricular systolic sound being accompanied by an arterial pulse perceptible in the radial arteries. But it sometimes happens that these sounds and silences succeed one another with perfect regularity over the cardiac area, and yet the radial pulse intermits; this is a sign of cardiac debility, and must be noted, that its cause may be inquired into. At other times the intermission extends to the cardiac action itself; or the cardiac sounds and silences, and also the radial pulse, may all be extremely irregular; and all these phenomena must be carefully noted, that their causes may be subsequently inquired into.



But there is still another form of irregularity in the cardiac sounds which may be a symptom of disease, but which may also occur in perfect health, and that is reduplication of these sounds. In its most extreme and rarest form we have, instead of two, four sounds,—two first and two second sounds; the two hearts acting separately and not simultaneously as usual. More usually, however, we have three sounds instead of two; and the rarest form of this is when the first sound is reduplicated, which is extremely seldom to be heard with any distinctness. A reduplication of the second, however, is a matter of common occurrence, not only in disease but even in health. It is heard as a *bruit de rappel* either at base, apex, or both. It may be vocalised by the syllables ta-ta, and closely resembles the sound of a hammer which strikes the anvil, rebounds and strikes again, remaining motionless. Reduplication of the second sound is of frequent occurrence in mitral constriction, due to the great pulmonary congestion, always present in these cases, which so increases the tension in the pulmonary artery as usually to accentuate, but sometimes so to accelerate the closure of its valve as to cause the pulmonary second to anticipate that of the aorta, and thus to reduplicate the sound. When reduplication takes place, closure of the valves must occur before the ventricular systole is finished, they are shut, therefore, in the face of a still advancing current, and in such circumstances, as you can readily suppose, forcible closure is not always possible and accentuation is frequently absent.\* Reduplications of the cardiac sounds in disease are

\* Ceradini states that the moment the blood pressure within the artery comes to equal that of the ventricle, the semilunar segments of the arterial valve fall together in their position of equilibrium with a snap that initiates the second sound, which is only strengthened, but not produced, by any of the events of the diastole. *Der Mechanismus der halbmondförmigen Herzklappen*, Leipzig, 1872, S. 62. If even in the normal condition we have a primary systolic closure of the semilunar valves, we can readily understand how this may take place earlier in one artery than in the other when the blood pressure within it is unusually high. This simplifies our idea of reduplication of the second sound, because it shows us how it may take place without any interference with the action of the heart, how it may even come and go with variations of the intra-arterial blood pressure, the action of both ventricles continuing to be both regular

often permanent ; in health they are more frequently fugacious, appearing one instant and disappearing the next. They are merely abnormal exaggerations of a phenomenon which, with care and appropriate appliances, may be detected in every one; reduplication of the first sound occurring at the end of the expiration or commencement of the inspiration ; reduplication of the second sound occurring at the end of the inspiration and commencement of the expiration. These normal reduplications depend upon the variations in the pressure produced by the respiratory movements at the origin of the arterial and venous systems.

The first sound becomes reduplicate because excess of blood-pressure in the heart retards the closure of the tricuspid valve ; and the second sound is reduplicated because excess of pressure in the aorta accelerates the occlusion of the aortic valves.\*

Within the cardiac area, as already remarked, murmurs may take the place of one or other of the normal sounds, or may occupy the time of one or other of the silences, or may even occupy the whole period of a cardiac pulsation, or any portion of it. Such murmurs may be of endocardiac or of exocardiac origin, and as endocardiac murmurs are most frequent, and are chiefly due to valvular lesions, it is a good rule to endeavour first of all to associate all murmurs with a valvular origin, and only after exhausting this hypothesis to proceed to determine the probabilities in favour of their extracardiac origin, or of their dependence upon some intracardiac cause apart from valvular lesion.

and synchronous. Nay, it even explains how we may have a reduplicated second when one of the arterial valves has been destroyed, a reduplicated pulmonary second accompanying free aortic regurgitation ; an unusual, but not unknown complication.

\* Potain, "Note sur le Dedoublement Normaux des Bruits du Cœur."—*L'Union Médicale*, 1866. In my own experience reduplicated first sounds have been comparatively rare. One of the most perfect instances of the kind was strikingly confirmative of Potain's view, inasmuch as impeded respiration, due to a distended ovarian cyst, was always accompanied by a reduplicated first, which invariably disappeared after tapping. This occurred repeatedly. There was no other cardiac abnormality.



Of all the signs of cardiac disease, murmurs are those most usually confided in, and yet they are really those of least value; first, because, as already remarked, exocardiac murmurs may simulate those of endocardiac origin; and, second, because murmurs truly of valvular origin may disappear temporarily or permanently.\* Thus we may have murmurs apparently of valvular origin which are really exocardiac in their origin; second, we may have murmurs truly of valvular origin, yet without permanent valvular lesion, which may disappear, leaving the heart uninjured; and, lastly, we may have murmurs truly of valvular origin which may disappear temporarily or even permanently, the valvular lesion still continuing. It is obvious, therefore, that murmurs cannot of themselves be accepted as certain indications of cardiac disease, even although we can positively connect them with the lesion of a definite valve, because that lesion may be temporary in its character, and wholly unconnected with actual disease of the valve affected. To this there is but one positive exception, and that refers to the auricular-systolic, the so-called presystolic murmur; though there is obviously a greater or less probability of any of the other valvular murmurs being also connected with actual disease of the valves affected. That probability, however, falls to be considered under the head of each separate valve; at present I have only to describe the acoustic phenomena audible on auscultation, to point out the means by which we determine whether these are valvular in their origin or not, and to state that no murmur, except the one already referred to, can ever be accepted as a definite sign of actual cardiac disease, but must be simply noted, to be afterwards duly considered along with the other information derived from inspection, palpation, and percussion, when we come to estimate the probabilities for or against the existence of any special lesion, for there is no royal road to diagnosis

\* "On the Variation and Vanishing of Cardiac Organic Valvular Murmurs," by W. R. Sanders, M.D., Professor of Pathology in the University of Edinburgh. —*Ed. Med. Journal*, Jan. 1869, p. 584.

any more than to anything else, that must be made, as Opie mixed his colours, "with brains."

When we hear, over any part of the area of cardiac dulness, any sound which differs from those sounds we have already taught ourselves to recognise as those audible in the normal condition of the heart, our first care must be to ascertain by careful examination at what part of the cardiac area this peculiar sound is most distinctly to be heard; this we term the position of maximum intensity. This position either coincides with one of the normal areas already described as the mitral, the tricuspid, the aortic, or the pulmonary area, or it does not. If the position of maximum intensity of this abnormal sound coincides with one or other of these normal areas, it most probably depends upon some lesion, temporary or permanent, of that valve whose normal sound is audible in that position, and which it either obscures, replaces, precedes, or follows. If it does not coincide with one or other of these normal areas, it is certainly not valvular in its origin, unless this position of maximum intensity be over the lower end of the sternum, or just to the left of the pulmonary area and in the same plane; and that any such murmur may be of valvular origin, it must be diastolic in the first position and systolic in the latter; the reasons for this being to be found in the anatomical relations of the parts connected with the valves at which such murmurs originate, and in the mode in which sound is conducted.

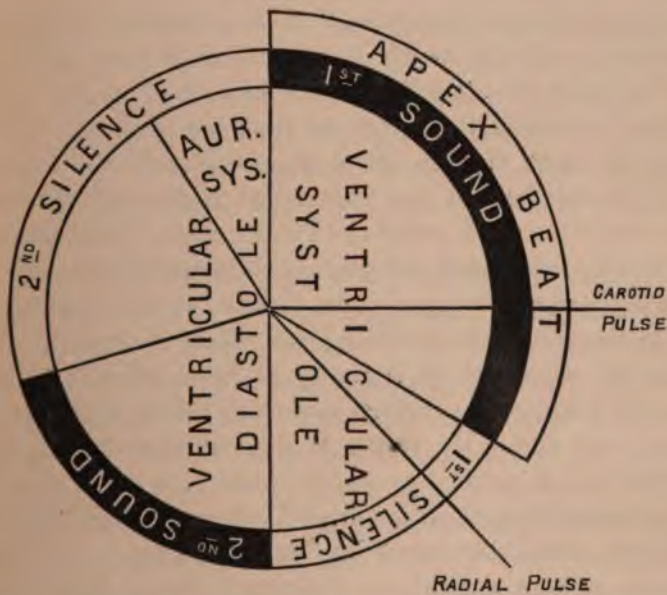
Our next care must be to determine the actual rhythm of the murmur or abnormal sound, that is, its positive relation to those several physiological acts which constitute a cardiac pulsation. That we may be able to do this, we must first of all have a clear conception of those physiological acts which make up what we call a cardiac pulsation; and, secondly, we must be able to recognise them. The accompanying figure (fig. 4, altered from Gairdner\*) represents diagrammatically the

\* To Dr Gairdner we owe the first attempt to represent a cardiac pulsation diagrammatically. This diagram, which is altered from his, is merely an attempt



several acts of a cardiac pulsation in the normal adult heart, in which a long sound precedes a short silence, followed by a short sound, and that again by a long silence. When the heart beats under 90 per minute, there is never any difficulty in making out the several relations of these sounds and silences, however slow the heart's action may be. When, however, the heart's action is over 90 per minute, it approaches

Fig. 4.



more or less to the uniform tic-tac of the infant's heart, and the more nearly this is the case the more equal are the two

to give more physiological accuracy to it. The apex beat and the two sounds are represented as occupying, as they do, appreciable intervals of time during that pulsation. The heart's action is represented as continuous and without actual pause or rest, though at times soundless, while the carotid pulse is placed in the position which Valentin says it ought to occupy in relation to the commencement of the apex beat, and the radial pulse is placed in more nearly its usual relation both to the carotid pulse and the apex beat. The foot pulse is nearly synchronous with the radial. According to Mr A. H. Garrod, the difference between the two, with a pulse beating 75 per minute, amounts to 0.0012 of a minute.

silences, and the greater is the difficulty experienced in determining what sound is long or first and what is short or second. To ascertain this with precision, we must either wait until rest and other agents have slowed the heart's action to 90 or fewer beats per minute, or we must depend upon our accuracy in the use of a double stethoscope, knowing that under all circumstances the first sound is always relatively long at the apex and short at the base, while the second is exactly the reverse; or we may determine what sound or murmur coincides with the apex beat or carotid pulse, that being certainly coincident with the ventricular systole; remembering that any reference to the radial pulse is irrelevant and misleading, as even in the normal condition it is separated from the apex beat by an interval which may amount to about one-sixth of a second, and that this interval may be increased in disease to the half, or even to the whole period of an entire cardiac pulsation.

Having determined the position of maximum intensity of the murmur, and ascertained its rhythm, we are prepared to state the nature of the lesion upon which it depends. All that we require to do is simply to remember that in the normal condition the auriculo-ventricular valves on both sides are closed during the systole of the ventricles, the arterial valves on both sides being opened; while during their diastole the reverse takes place, the arterial valves are closed and the auriculo-ventricular valves are opened. Thus a murmur, whose position of maximum intensity is in the mitral area, depends upon some defect in the mitral valve: if it be synchronous with the systole of the ventricles, it depends upon some defect in its closure, and is therefore a murmur of regurgitation backwards into the auricle; but if it be synchronous with the diastole of the ventricles, it depends upon some obstruction to the flow of the blood through the open mitral valve, and is therefore a murmur of obstruction. The first of these murmurs runs off from the apex beat, and more or less completely replaces the normal first sound. The second murmur accompanies or follows the second sound, but in no



respect interferes with its production. But there is a third murmur, or portion of a murmur—for the murmur may be continuous—which follows the second sound by an appreciable interval, and whose position of maximum intensity is not only placed in the mitral area, but which is but little audible out of that area,—this murmur immediately precedes and runs up to the first sound (is therefore presystolic); but a glance at the diagram (fig. 4) shows us, what we indeed already know, that the systole of the ventricles is immediately preceded by the systole of the auricles, and that what we term presystolic is truly auricular-systolic in its rhythm. This murmur is not only a murmur of obstruction, but a murmur of obstruction to direct cardiac action, and, like all such murmurs, is always rough. It is the only generally recognised cardiac murmur which is invariably associated with actual disease of the valves affected.

These three murmurs may exist separately, as occasionally they do, or any two of them may coexist; and sometimes the whole three are present, and then we have a murmur audible in the mitral area, and running through the whole period of cardiac action, the first sound being either much altered or entirely replaced; while the second sound, though not always audible at the apex, always exists, the pulmonary second being greatly accentuated, unless two second sounds are heard separately, the one immediately following the other, constituting what I have already mentioned as reduplication of the second sound, in which case the accentuation of the pulmonary second is always modified, and generally absent. These murmurs, though most common on the left side, may also be heard over the right heart, a systolic tricuspid murmur being very frequent, a presystolic tricuspid being somewhat rare, while a diastolic tricuspid is possible, but, so far as I know, hitherto unrecorded. And in saying this it is obvious that I refer to a diastolic murmur over any part of the right auricle or ventricle, both the aortic and pulmonary second sounds continuing audible, and with entire absence of any symptom or sign of

aortic regurgitation. This is a most important point, because it often happens that a diastolic murmur, originating in defective closure of the aortic valves, is heard loudest over the lowest part of the sternum, being conducted downwards partly by the descending current of the blood, and partly by the peculiar resonance of the sternum. We shall afterwards see that a diastolic murmur, of purely aortic origin, may have its position of maximum intensity over the lower part of the sternum—the tricuspid area—or in the mitral area, and that this is said to depend upon the segment of the valve affected.

At the base of the heart the audible murmurs are apparently less complicated than at the apex, because here we have no presystolic murmur, the only possible murmurs being systolic and diastolic. But the causes of these are so various, that the pulmonary region has somewhat deservedly received the name of the region of romance, from the various theories propounded in explanation of the murmurs occurring there; while the physical causes of aortic murmurs, and the physical relations of the aorta itself, are so complicated, and so efficiently modify the conduction of these murmurs, that though it is quite usual to say that a systolic murmur, having its position of maximum intensity at the sternal end of the second right costal cartilage, is due to aortic obstruction, yet this is by no means always the case; and in like manner, though a diastolic murmur, audible in that position, is most probably due to aortic regurgitation, yet its absence from that situation is no proof of the absence of regurgitation, because the murmur due to regurgitation may be only audible beneath the level of the aortic valves.

The important thing for us to remember is, that in noting the characteristics of a murmur, the chief points to be determined are, *first*, the position of maximum intensity, as probably indicating the valve affected; *second*, the rhythm of the murmur, as indicating whether it is systolic or diastolic; and, *third*, whether the valve sound normally heard in any given position is wholly replaced by the murmur, or only altered in duration and character. We must also remember

that even the existence of a well-defined murmur is not by any means a certain indication of actual valvular disease, any more than is the absence of any murmur to be regarded as a sure proof of the non-existence of valvular disease. The characteristics of a murmur, and its relations to the normal sounds and silences, must merely be noted and afterwards duly considered in connection with the other physical signs already commented on.

Endocardiac murmurs of other than valvular origin are occasionally, though rarely, observed. Sometimes these are due to congenital deficiency of some part of the septum between the two hearts; and in that case they only intensify those valvular murmurs which almost invariably coexist, and extend their area of audibility; or where no valvular lesion coexists, as in those endocardiac murmurs, the occurrence of which is very problematical and must certainly be rare, and which are said to depend upon the presence of some vibrating substance, as a flake of lymph, upon one of the *cordæ tendineæ*, in an otherwise healthy and normal heart, the position of maximum intensity and the rhythm may possibly be the same as those of truly valvular origin, from which they ought to be readily differentiated by a due consideration of the other physical signs; that is, by a judicious reference to those physical facts which, as I have already shown, indicate actual obstruction to the onward flow of the blood, and which point out its existence with a certainty, the attainment of which is exactly proportionate to our skill in interpreting these signs. Exocardiac murmurs frequently simulate in their timbre those of endocardiac origin, and when they also coincide in rhythm with the systole or diastole of the heart, it may be difficult to distinguish such a murmur from an endocardiac one. As, however, exocardiac murmurs depend chiefly upon the friction of the roughened visceral and parietal portions of the pericardium upon one other, or of the pericardium upon the pleura, and as, unless under very exceptional circumstances, this friction is always greatest over the centre of the heart, and neither at its

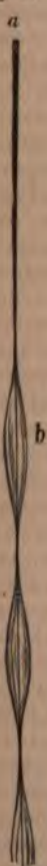


apex nor exactly at its base, we have a pretty safe indication of the exocardiac origin of the bruit in the non-coincidence of its position of maximum intensity, with the usual situations of that position in the case of murmurs which are of valvular origin. We have also the localisation, more or less complete, of the bruit, which never passes to any distance from the area over which it is most audible, and which is not propagated by any blood-current, but simply radiates equally all round the position where it originates, usually possessing a distinctly superficial character; and we have the entire absence of any pulmonary accentuation, or of any physiological indication of obstructed circulation. The sound produced also, as a rule, resembles the crackling of parchment, the creaking of new leather, or a grating or mere rubbing sound, of greater or less intensity, and only very rarely in any respect simulates the soft blowing murmur of a valvular lesion. Whatever may be its timbre or its rhythm, it must, like all and every other sound heard over the cardiac area, be carefully noted as to its character, position of maximum intensity, rhythm, and direction in which it is propagated, to be afterwards submitted, along with the other facts ascertained, to a careful and discriminating scrutiny. The same may be said of those murmurs which are audible in various positions out of the cardiac area, and which owe their origin to the passage of the blood through the vessels. Such murmurs are sometimes heard in the arteries and sometimes in the veins. They are chiefly systolic, but sometimes diastolic in rhythm. In the arteries these murmurs may be strictly localised, or may be audible over every part of the arterial system. In the veins, as a rule, they are audible everywhere, provided certain conditions are complied with, though they are only permanently present in certain parts of the body where these conditions naturally exist. Such intravascular murmurs are of the most various significance, and can only be most briefly referred to now. Sometimes they signify serious lesion of the vessels themselves; at others, nothing worse than some slight

deformity of the chest, the result of rickets; and at still other times, only an alteration in the condition of the blood itself, which may arise from various causes.

I cannot conclude this lecture without a cursory reference to the manner in which these murmurs are believed to be produced, because this is a subject in regard to which practical experiment may yet become a most fruitful source of improvement in diagnosis. The crude ideas of our forefathers, which

Fig. 5.



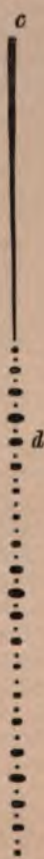
culminated in the notion that murmurs are produced by the friction of the blood-current on its containing walls—an idea which the researches of Poiseulle and others have set aside, by showing that no such friction exists—have been replaced by a recurrence to the theory, first started by Corrigan, that murmurs occurring in the course of the circulation are due to the vibrations induced by eddies in the blood-column itself, a theory which has been reduced to scientific simplicity by M. A. Chaveau,\* who has with great

\* *Comptes Rendus de l'Académie des Sciences*, 1858—"Toute veine fluide est le siège de vibrations susceptibles de produire des sons, vibrations qui ébranlent aussi l'orifice d'écoulement de la veine"—p. 841. The intravascular murmurs of anæmia are due "aux vibrations de la veine fluide intravasculaire, vibrations qui se produisent toujours quand le sang pénètre avec une force suffisante dans une partie réellement ou comparative-ment dilatée de l'appareil circulatoire"—p. 933.

Perhaps the nature of this fluid vein may be more readily understood by a short account of the conditions under which it is known to be formed. When fluid escapes through a perforation in a thin plate forming the bottom of an open vessel kept constantly full, Torricelli supposed that

the efflux could be ascertained by this theorem:— $Q = ts \sqrt{2gh}$ , in which  $Q$  represents the outflow,  $t$  the time involved,  $s$  the sectional area of the orifice,  $g$  the acceleration of gravity, and  $h$  the height of the surface above the orifice.

Fig. 6.





probability referred the causation of all murmurs, whether intracardiac or intravascular, to the sonorous vibrations of fluid veins, such as have been observed by Savart, and which are capable of being transferred, as Marey has experimentally shown, to the surrounding fluid within which the vein may be

The actual outflow when measured is, however, always about one-third less than that given by calculation, and the discrepancy has been found to be due to the fact that the velocity on which the calculation is based is only true of the central part of the jet, the outer portion being retarded by the interference of the currents converging from all sides of the vessel. The convergence of these currents also causes the jet to taper off to  $\frac{1}{6}$  of its original dimensions at a distance from the orifice of one-half its diameter, and if the sectional area of this narrow portion, the *vena contracta* as it is termed, is substituted for that of the original orifice, the actual outflow is then correctly expressed by Torricelli's theorem. Up to this point the jet has been clear and pellucid, but now, probably from the conflict between the tendency to diverge inherent in the outer currents and the attractions of cohesion and gravity, the jet loses its transparency and breaks up into those vibrating fluid veins the phenomena connected with which have been thus described by Felix Savart. The part of the vein *a b*, fig. 5, is steady and limpid, presenting the appearance of a solid rod, which obstructs vision and wets the finger when passed through it. This decreases in diameter till it reaches a point (just above *b*) of maximum contraction, beyond which it is turbid and unsteady, marked by periodic swellings and contractions, does not wet the finger when passed through it, and does not obstruct vision, even though the fluid be mercury. At *b*, in fact, the liquid is no longer continuous, but resolves itself into a series of liquid spherules, which have an appearance of turbid continuity, from their rapid succession never permitting the primary impression wholly to fade from the retina still succeeded by a second. Fig. 6 represents this fluid vein illuminated by an electric flash, when the drops of which it consists are seen as it were motionless in the air, and the cause of the periodic swellings and contractions are at once apparent; for these spheroidal drops, when first detached, have their long axis vertical; when abandoned to their own molecular forces they seek to become spheres, and, like a pendulum in motion seeking to return to rest, the contraction goes too far, and they become flattened spheroids, which again elongate vertically; hence the appearance of alternate swellings and contractions. Savart traced the production of these pulsations to the orifice through which the vein passed, but did not regard them as the result of friction. Under moderate pressure they succeed each other sufficiently rapidly to produce a feeble musical note, the pitch of which may be fixed by allowing the drops to fall upon a stretched membrane.—*Vide Tyndall On Sound*, London, 1869, p. 244, &c. By attaching tubes of various forms to the original orifice, the formation of a fluid vein may be completely prevented, and the discharge may be even increased beyond the quantity calculated by Torricelli. It seems probable that the relations of the several parts of the circulatory system to one another are adjusted in some such manner, so that it is only when disease has altered the normal relations that we have the formations of sonorous fluid veins.

produced, and to the walls containing it. The roughness of the murmur and the tactile perceptibility of the vibrations are in some degree proportionate to the force exerted in producing this sonorous vein, while its musical character must of course depend upon the rapidity of these vibrations, as well as on the physical structure of the parts to which these vibrations are communicated.

But the mode in which the production and conduction of their sonorous vibrations may be modified by evident or imperceptible alterations in the heart's action itself, or by alterations in the anatomical relations of the parts concerned, produced by changes in the position of the patient himself, are problems which yet remain to be defined, and a careful experimental investigation of those problems will certainly prove to be of the utmost importance in the accurate diagnosis of cardiac and vascular diseases.

## LECTURE II.

## ON INCOMPETENCY OF THE AORTIC VALVES, WITH SPECIAL REFERENCE TO THE DIAGNOSTIC SIGNIFICATION OF THE MURMURS.

GENTLEMEN,—There is no disease of the heart, of common occurrence, which possesses so many elements of interest as aortic incompetency, whether we regard its pathology in relation to the various causes which may give rise to it, to the concomitant morbid alterations in the aorta, in the heart itself, and in the circulation, which are so various and sometimes so diverse; or whether we regard its diagnosis, which, to be of value, involves a careful discrimination of the causes of the incompetence, and of the consecutive changes in the heart itself, upon a due appreciation of which both the prognosis and the treatment largely depend.

In aortic incompetence, as well as in all cardiac diseases, all our information is derived from the state of the pulse, and from the inspection, palpation, percussion, and auscultation of the patient; and a due consideration of these phenomena enables us to predict with considerable certainty the exact condition of the aortic valves, and to determine the degree in which the persistence of this condition has already influenced the walls of the heart as well as the circulation generally. Confining our attention for the present to the auscultation of the patient, you are aware that the murmur indicative of aortic incompetence is a diastolic murmur of a more or less soft and blowing character, audible, as is usually said, at the base of



the heart. But this is by no means always the case. Sometimes the murmur is only audible, and that but faintly, just below the aortic valves, at midsternum, a little beneath the level of the third rib. Very rarely, it is loudest at the left apex;\* very frequently it is loudest and sometimes only to be heard—except in the arteries—at the ensiform cartilage, or more nearly in the position of the right apex; at times it is loud and distinctly audible over every part of the cardiac area, and then it is also audible in every part of the arterial system to which the stethoscope has access; while at still other times it is inaudible above the level of the aortic valves, and is not perceptible in any artery. I have already mentioned† that murmurs are produced by the formation at any point of a liquid vein in the blood-current, and an instant's reflection will show us that the same cause which is capable of producing a liquid vein at the aortic orifice during the diastole of the heart must in most instances be equally potent during its systole; hence we have in such cases almost always a double murmur *bruit de va et vien*, a see-saw murmur, of which the first or systolic portion is usually rough, the second, or diastolic portion, always soft and blowing, though sometimes the one and sometimes the other may be loudest; while the propagation of the systolic portion of the murmur is subject to apparently similar vagaries with that of the diastolic portion, being at one time audible over all the cardiac area, at another time only audible at and above the aortic orifice, and again chiefly audible only in the arteries. The term double aortic lesion is therefore a common and by no means inapplicable designation for that affection of which incompetence of the aortic valves forms always the most distinctive and rarely the sole important feature. Nor are the variations in the manner in which the

\* This position of maximum intensity of the murmur of aortic incompetence is said to be an indication of rupture or disease specially affecting the posterior or mitral segment of the semilunar valve.—*Vide* Dr Balthazar Foster's *Essays on Clinical Medicine*, London, Churchill, 1874, p. 121. But most observers are agreed that this idea is highly problematical.

† *Vide antea*, p. 43.

constituents of this double murmur are propagated mere vagaries in the conduction of sound; they are important facts depending upon physical causes, and convey most momentous information to the understanding of the physician whose ear perceives them.

The following cases have been selected for the purpose of exhibiting the chief important varieties of aortic regurgitation, and the manner in which the condition of the valves in each variety influences the production and the propagation of the murmurs:—

CASE I.—Janet M'Gowan,\* aged 22, admitted, 25th March 1874, to bed 2, Ward XIII., complaining of subacute wandering pains in her joints. This attack commenced twelve months ago, and was at first accompanied by great pain and some swelling in the region of the heart, for which a fly-blister was prescribed, after this the swelling disappeared and the pain subsided, but it has not altogether ceased. Three years and a half ago she had an attack of rheumatic fever, but apart from that has always enjoyed good health, excepting suffering from measles and scarlet fever when a child. The patient is well developed; expression natural; integuments moist; temperature  $98.6^{\circ}$ ; limbs somewhat wasted; joints noways deformed, but occasionally painful, chiefly the shoulder and elbow joints; no anasarca; arteries and veins normal on inspection; pulse 73, slightly jerking, but this is not perceptibly increased on elevating the arm at right angles to the body as the patient lies in bed. On percussion, the cardiac dulness at one inch to the left of the sternum extends vertically from the upper border of the third rib till it meets with the tympanitic resonance of the stomach, and extends in the line of the fourth rib from three-quarters of an inch to the right of the sternum transversely across the chest for a distance of four inches and three-quarters. The heart's apex beats firm and full between the fifth and sixth ribs, two inches and three-quarters to the left of the sternum. On auscultation in the mitral area, the first sound

\* Condensed from the notes of Mr J. H. Clarke, clinical clerk.

is heard loud, thumping, and accompanied by a systolic murmur ; no diastolic sound or murmur being audible. In the tricuspid area the first sound is loud, distinct, still somewhat thumping, and accompanied by a systolic murmur, followed by an equally distinct diastolic one. Above the fourth rib the thumping character of the first sound is lost, while the systolic murmur increases in intensity. In the aortic area neither systolic nor diastolic sound is audible, but instead there is a very loud and distinct double murmur, the systolic portion of which is extremely rough, and propagated with great distinctness into the carotid arteries, the diastolic murmur being in them inaudible. In the pulmonary area a double murmur is heard, the systolic portion of which completely replaces the first sound, while the diastolic portion partially obscures an accentuated second sound. Her respirations are 32 in a minute; no cough, and no dyspnoea, except on exertion, and then not to any great amount; pulmonary physical signs normal; nervous system normal; appetite defective; bowels regular; urine normal, specific gravity 1015; menstruation regular till twelve months ago; since then irregular. On 15th May it is noted that a well-marked presystolic murmur has become developed, and this continued till the patient was sent to the Convalescent Hospital on 20th May, completely relieved of all her rheumatic symptoms. This patient, you will observe, came under treatment solely for her subacute rheumatic affection, and though labouring under serious cardiac disease, which will undoubtedly shorten her days, this had been so completely compensated as to be altogether mute. The dilatation and hypertrophy of the left ventricle sufficiently counterbalanced each other in such a manner as to compensate the existing regurgitation through the incompetent aortic valves. For the present I omit as irrelevant all reference to the state of the mitral valve, and shall direct your attention solely to the aortic murmurs and their mode of propagation. You will observe that the systolic murmur, more or less audible over the whole cardiac area, has its position of maximum intensity in the aortic area, and is



therefore clearly of aortic origin. It depends, therefore, upon some abnormal alteration of the aortic orifice capable of producing a fluid vein, and so giving rise to an audible murmur. Now, the essential element in the production of a fluid vein is some abnormal relation between the part where the blood comes from and that into which it flows—in this case, the ventricle and the aorta—and to produce a systolic fluid vein the latter must be really or comparatively dilated. There is in this case no evidence of any real dilatation of the aorta—no increase of dulness over the upper part of the sternum, no pulsation in the tracheal fossa. Have we any reason to suspect the existence of a comparative dilatation, that is, of an obstruction at the orifice, and what is its nature? The answer to this question is easy. From the history of this patient we have every reason to believe that she has had pericardiac inflammation, as proved by the swelling and pain in the precordial region in the early period of her rheumatic attack; while the thumping first sound and subsequently developed presystolic murmur, along with the more or less constant cardiac pain, conclusively prove that this has been associated with a more or less chronic endocarditis, which has stiffened and deformed the mitral valve. It is natural to suppose that a similar effect has been produced upon the aortic semilunar valves, the result of which has been that they now obstruct the onward current of the blood, and so produce a relative dilatation of the aorta, and give rise to a systolic fluid vein and concomitant murmur, rough in proportion to the ventricular force employed in expelling the blood, and to the degree of obstruction existing, which, in accordance with the laws of sound, would be carried most distinctly onwards with the blood-current into the carotids, while it would be also propagated with greater or less distinctness, according to circumstances, in other directions. The history of this case is therefore in complete accordance with this portion of the auscultatory phenomena, and renders this explanation probable in the very highest degree. But if the aortic semilunar valves are so thickened and crumpled as to

produce obstruction to the onward flow of the blood, they can hardly be expected to be also competent. They may be so, but only very rarely. Most usually when in such a condition they are more or less incompetent; and then the same cause which gave rise to a fluid vein and a murmur during the systolic outflow of the blood from the ventricle must likewise produce a fluid vein, and consequently a murmur, during the diastolic regurgitation of the blood from the aorta into the ventricle; the diastolic murmur being softer than the systolic one, because the forces expelling the blood from the artery are so much less powerful than the contractile force of the left ventricle; and this has evidently been the case in this instance. We are scarcely yet in a position to state in every case why a diastolic murmur originating at the orifice of the aorta should at one time be heard loudest above that part, as in the so-called aortic area, and at another lower down the sternum. At present we can only state it as a fact, and indicate the strong probability there is that it will ultimately be found to be connected with the nature of the obstruction; and the comparative readiness with which the vibrations of the fluid vein are propagated outwards through the walls of the vessel, on the one hand, or downwards with the current of the blood, on the other. In all probability the greater the obstruction, and the more vibratile its structure, either in itself or in its connection with the arterial walls, the more the murmur will be localised in the aortic area; while the less the obstruction, and the less vibratile its structure or connections, the more the murmur will tend to be propagated downwards in the direction of the blood-current, or possibly it may be solely produced at that part of the heart on which the vein impinges, propagation in other directions ensuing in all cases in accordance with the known laws of sound. These reasons have at least probability on their side, and you will find that the more we examine into the various forms of aortic incompetence, the greater will this probability appear. At present we are dealing with a case in which both the systolic and

diastolic murmurs are most distinct in the aortic area. The incompetence of the aortic valves is not great—1st, Because the apex beats in its normal plane, though a little outside of its normal position; the heart has more of the purse shape usual in mitral disease than of the conoid form, due to dilatation of the left ventricle, which is so common in aortic incompetence. 2d, Because there is but little of that jerking character to be felt in the pulse, or seen in the carotid and brachial arteries, which is so distinctive of free regurgitation through the aortic valves. The absence of this is of course not due in this case, as it sometimes is, to debility of the ventricular walls, because the apex beat of this heart is firm and forcible, but obviously to the absence of any great dilatation of the left ventricle, and, as an ultimate cause, to the absence of any great aortic incompetence. 3d, Because the diastolic murmur is quite inaudible in the carotid as well as in the crural arteries, while it is so loud at the base of the heart. We shall presently see that a diastolic murmur may be audible in the arteries and inaudible at the base of the heart, and that in this case it is associated with all the signs and symptoms of great valvular incompetence; while in our present case the signs and symptoms are the very reverse. For these reasons, therefore, there are the strongest grounds for believing that in this case we have to do with—confining our attention to the aortic valves—a stiffened and somewhat shrivelled condition of these valves due to rheumatism. These valves may of course be also atheromatous, or covered with dense vegetations, possibly both, and are only slightly incompetent, the mitral lesion being comparatively unimportant, though its coexistence necessarily renders the prognosis more grave.

The following example exhibits how these murmurs of aortic incompetence vary even in cases which are otherwise similar, showing, also, how all such variation is probably due to slight alteration in the physical causes, the signs of which we only want a larger and more accurate experience to enable us to interpret correctly.

CASE II.—Mary Anderson,\* married, a laundress, aged 31, admitted to Ward XIII., 3d December 1872, complaining of severe pain in the region of the heart, a choking sensation in the throat, a troublesome cough and spit, and complete loss of sleep. Nine years ago she suffered from a sharp attack of rheumatic fever, lasting eight weeks, six of which were passed in hospital. At this time she had severe pain in the region of the heart, both in front and behind, accompanied by palpitation. A fortnight after dismissal she was able to return to her work. A similar but less severe attack has recurred every winter since that time. The present attack commenced eight weeks ago with severe pain in the cardiac region, with swelling of the legs, choking sensation in the throat, which did not interfere with swallowing; atonic dyspepsia, relieved temporarily by occasional vomiting after food; and gradual diminution in the amount of urine passed. Patient's family history is good. She has two children, both alive and healthy. Her face is somewhat livid, her fingers slightly clubbed, her legs œdematous, the joints normal, temperature 99°, sensation and intelligence normal; has slept well the two nights following admission; pulse 112, irregular, somewhat forcible, and felt to be distinctly jerking on elevating the arm at right angles to the body as she lies in bed. Considerable pulsation is visible at the root of the neck, both in the carotids and in the tracheal fossa. No venous pulsation is observable. The apex beats somewhat forcibly between the sixth and seventh ribs, about three and a half inches from the left edge of the sternum. In the parasternal line, one inch from the left edge of the sternum, dulness begins at the upper border of the second rib, and extends down to the liver dulness. Transverse dulness in the line of the fourth rib commences at three-quarters of an inch to the right of the sternum, and extends across for four and three-quarter inches. On listening over the apex beat, a slight thump is heard, forming as it were the first portion of the first sound, which is obscured by a bruit. The second sound in

\* From the notes of Mr O'Connor, clinical clerk.

this area is replaced by a soft diastolic murmur. In the tricuspid area, the first sound is quite audible, the obscuring bruit being, however, louder than in the mitral area, the diastolic murmur being still soft, but also more distinct than in the mitral area. In the aortic area, the first sound, audible in both the mitral and tricuspid areas, is completely replaced by the systolic bruit, which is loud and rough, and attains its maximum intensity in this position; the second sound is wholly wanting, being replaced by a soft, low, but distinct diastolic murmur, the position of maximum intensity of which is at midsternum, just below the fourth rib. In the pulmonary area, the first sound is wholly replaced by a loud systolic murmur, while the second is heard loudly accentuated, but somewhat obscured by the diastolic murmur propagated across from the aortic area. In the carotid and femoral arteries, a loud systolic murmur alone is heard, the diastolic murmur being inaudible. Her respirations are 32 in a minute. She has a sharp, harsh cough, accompanied by slight watery expectoration. Pulmonary percussion everywhere normal. Auscultation reveals crepitation over both lungs anteriorly; posteriorly, this is mingled with occasional rhonchi. Tongue clean but red and raw-looking at its tip. There is great craving for food, some flatulent distension of the stomach, especially after eating; bowels irregular; liver dulness normal. Patient has not menstruated for twelve months. Her urine varies from 36 to 52 oz., of a clear straw colour, with a trace of albumen on admission, which has since disappeared.

*Diagnosis.*—The history of this patient is that of rheumatic fever, accompanied by an affection of the heart, which inspection, palpation, and percussion agree in showing has resulted in dilatation of the heart, particularly of its right auricle and left ventricle, with some hypertrophy of the latter. The slight thump forming the first portion of the first sound is well known to be the last remaining vestige of a presystolic murmur, quite as distinctive to an ear accustomed to recognise it as a well-marked auricular-systolic murmur, and equally with it a sign



upon which it is safe to rely with the utmost confidence as significant of constriction of the auriculo-ventricular opening. But mitral stenosis is one of the most common results of a cardiac rheumatic affection, and of itself is capable of explaining the pulmonary congestion and œdema, revealed by the cough, the watery expectoration, and the accentuated pulmonary second. In the absence of any serious implication of the right side of the heart—which cannot exist because a tricuspid first sound is distinctly audible, and also because there is not any marked pulsation either at the lower end of the sternum or in the jugular veins—this stenosis seems insufficient to account for the great interference with the systemic circulation shown by the livid countenance, œdema of the lower extremities, slight albuminuria, and flatulent dyspepsia; while the very considerable dilatation of the left ventricle which undoubtedly exists, would certainly be unusual as the result of mitral constriction alone. But, on the other hand, we have in the loud systolic murmur, with its position of maximum intensity in the aortic area, and which is distinctly propagated into the arteries, sufficient proof that the same disease which has deformed the mitral valve has also altered, thickened, and stiffened the aortic valves, while the diastolic murmur which has replaced the aortic second sound shows that the crumpling and stiffening of the aortic valve, which is great enough to retain them as a considerable and permanent obstacle to the egress of the blood, is also sufficient to prevent them closing with that tension necessary to the production of a diastolic sound, and has rendered them incompetent. This incompetence, however, though sufficient to produce a murmur at and below the situation of the aortic valves, is so trifling as to have but a very slight influence on the arterial circulation, and is therefore not audible in the carotid arteries, and is only carried up to the so-called aortic area, by propagation through the aorta itself and the bones of the sternum, while it is carried downwards to a greater extent, as in conveying it in that direction the influence of the blood-current coincides with that

of the sternum. So trifling an incompetence of the aortic valves would, in ordinary circumstances, be readily compensated, but in the present case it greatly intensifies the evil influence of the existing mitral constriction, and both together form an obstruction to the onward flow of the blood, quite sufficient to account for all the serious symptoms which exist, and also to render the prognosis very grave in this patient, who has been so greatly enfeebled by her repeated rheumatic attacks. The patient was treated with nourishing unstimulating diet—with half-ounce doses of the infusion of digitalis and five grains of carbonate of ammonia thrice a day, and fifteen grains of chloral at bed-time, to allay her cough and cardiac pain and procure sleep. At first she progressed favourably, but towards the end of December (21st) she began to complain of pains in her joints, increased œdema of her legs, and considerable pain over her stomach and liver, the latter now commencing to enlarge and become tender to touch.

She now had given her ten minims of the tincture of digitalis, with five of the liquor arsenicalis and of the tincture of the perchloride of iron, thrice a day. She gradually became worse, however, and in the beginning of January diarrhœa set in. This was kept in check by appropriate treatment, but the pain in the chest, the breathlessness, and pain over the liver continued, and she died at 1 A.M. on the 15th of January 1873.

*Autopsy.\**—Jan. 17. Heart considerably enlarged; mitral valve constricted, admitting only one finger, aortic valves atheromatous, the cusps covered with vegetations. Several hæmorrhagic infarctions in the lungs, which are otherwise healthy. Liver weighs 4 lbs. 14 oz., is of firm consistence, slightly hypertrophied, and exhibits all the characteristics of extreme chronic congestion. Spleen weighs 11 oz., firm and congested. Kidneys are slightly congested, and exhibit several depressions, the results of previous infarctions; the right weighs 6½ oz., and the left 5½ oz. Other results unimportant.

\* From the Pathological Records of the Royal Infirmary. Weight of heart not recorded.

These two cases, then, exhibit the character of the murmurs, and the manner of their propagation in those whose aortic valves are somewhat crumpled, thickened, and atheromatous, mainly as the result of rheumatism. Such valves cannot move out of the way, they therefore present a considerable obstacle to the egress of the blood, and this results in the production of a loud systolic murmur, always audible without compression in the carotid arteries, and dying gradually off within the arteries as the distance from the heart increases. As these valves move but little out of the way of the cardiac systolic current, they have no distance to fall during the cardiac diastole; therefore, so far as they do close at this time, this closure is not forcible; and the cusps being no longer membranous, every source of tensile vibration is almost completely cut off, and there is no trace of an aortic second sound to be heard. We must be careful to remember that the entire absence of a second sound in such cases does not arise from the completeness of the destruction of the valves, because they are still present, and frequently occupy even a larger space of the aortic orifice than they do in other cases where an aortic sound is partly audible; neither is it because the second sound is obscured by the diastolic murmur. This might be the case, as when the valves are in a different condition a second sound and a diastolic murmur, originating at the same orifice, are, at least occasionally, to be heard together; in cases such as the present, however, the absence of a second sound is evidently solely to be attributed to the immobility and absence of membranous elasticity in the valves themselves. On the other hand, the deformity of the aortic valves prevents them from closing perfectly, and permits of regurgitation proportionate to that imperfection; as this is seldom great, the diastolic murmur to which it gives rise, however loud it may be, is never propagated into the carotid arteries. It may be heard all over the cardiac area, propagated chiefly through the bones, the sternum acting as a sounding-board; but it is chiefly heard about midsternum, just below the fourth rib, and is

under all circumstances propagated downwards with most distinctness. The essential characteristics of the physical signs quoad the murmurs being in such cases the loudness of the systolic murmur in the aortic area and its ready propagation into the carotids; while the position of maximum intensity of the diastolic murmur is just over the aortic valves, or immediately below them, and it is never propagated into the carotids, or only in the faintest possible manner, such faint propagation being always an indication of a regurgitation greater than usual.

The next case which I shall relate presents phenomena which are exactly the reverse of these, and indicate of course a different character of lesion.

CASE III.—James Bailey,\* aged 35, unmarried, a labourer, residing in Leith, was admitted to bed 2, Ward V., on 19th February 1874, complaining of a severe cold, continuous cough, and pain in the epigastrium. The patient says that he has been short of breath as long as he can remember, and he thinks this has got worse as he grew older. Twenty years ago he had fever (probably typhus), for which he was treated in this infirmary. Nine years ago he was under treatment in this infirmary for one month on account of rheumatic fever; during this illness he was delirious for two or three nights. About three years ago he was again in this infirmary for a less severe attack of rheumatism. He has always been well fed, comfortably housed and clothed, but his occupation necessitates constant exposure to all kinds of weather. About three weeks ago he was more than usually exposed to severe weather, and except his dyspnœa, all his ailments date from this time. Patient is fairly developed, height 5 feet, weight 8 stone 9 lbs., muscularity good. He usually sleeps on his left side, but is often obliged to sit up in bed for a time on account of his dyspnœa. Expression of his face is normal, integument normal, temperature 98°, limbs and joints natural; pulse 75 per minute, full, jerking, and delayed about half a pulsation

\* From the notes of Mr A. Field, clinical clerk.

behind the apex beat; very evident and distinct pulsation is to be seen in both carotids and in both brachials. On inspection, the apex is seen to beat with tolerable distinctness in the seventh intercostal space, five inches to the left of the xyphoid cartilage; from this an undulatory pulsation is diffused up to the fourth rib, the most remarkable part of which is a systolic depression, greatest between the fifth and sixth ribs. In the scrobiculus cordis, a slight pulsation is distinctly visible, and this is seen to be communicated to the entire hepatic region. On palpation the diffused apex beat is felt to extend over some inches; it, as well as the whole left ventricle, communicates a heaving but not forcible impulse to the hand. In the parasternal line, dulness commences at the third interspace and extends downwards to the liver dulness; at the level of the fourth rib, the transverse dulness extends from half an inch to the right of the sternum to half an inch beyond the left nipple, a distance of six inches and a half. On auscultation in the mitral area, the first sound is obscurely heard and of an impure quality, but without distinct murmur; the second sound is heard obscured by a diastolic murmur. In the tricuspid area, the first sound is much more clear and distinct than over the mitral area, no second sound is audible, it being completely replaced by a loud and well-marked but soft diastolic murmur. In the aortic area, the first sound is obscurely heard, much as at the apex, and without evident murmur; the second sound is replaced by a distinct diastolic murmur. In the pulmonary area, the first sound is impure; the second sound accentuated, but obscured by the diastolic murmur. The diastolic murmur is heard over every part of the chest, but is loudest over the sternum, beneath the aortic valves; it is even audible over the chest posteriorly: in the carotid and femoral arteries, a well-marked and distinct double murmur is readily heard. The percussion of the lungs is everywhere normal, but on auscultation a good many rhonchi and crepitant rattles are heard distributed over them. Liver dulness is normal; and except some flatulent atonic dyspepsia,



there is nothing else of importance about the patient. Bailey continued gradually to improve under treatment till 14th April when he was discharged. Since then he has written from Ireland to say that he has given up all remedies, and feels quite well. The treatment consisted mainly in the continuous and regulated administration of digitalis, at first in the form of  $\text{℥ss}$ . doses of the infusion, with five grains of carbonate of ammonia thrice a day; latterly the tincture was given with iron and arsenic. Apart from the lung symptoms, which were mainly due to a severe catarrh, aggravated by the continuous pulmonic congestion maintained by the aortic insufficiency, the remarkable facts in this case were, 1st, The great dilatation and hypertrophy of the left ventricle, revealed by the low situation of the true apex, which beat beneath the edge of the eighth rib; the general slow heaving impulse of the ventricle, and the systolic dimpling of the surface over the left ventricle, the latter being evidently due to displacement of the lung, and the pressure of the atmosphere upon the walls of the chest during the systolic recession of the enlarged ventricle. 2d, The absence of any special implication of the right heart, notwithstanding the great amount of disease on the left side. 3d, The impurity of the mitral sound, obviously depending upon some slight rheumatic alteration of the mitral valve. 4th, The absence of a marked systolic murmur from the aortic area. 5th, The loud diastolic murmur, audible over all the cardiac area, beneath the level of the aortic orifice. 6th, The markedly distinct character of both murmurs in all the arteries, and specially in the carotid and femoral arteries. The absence of any distinct systolic murmur from the aortic area indicates the absence of any well-marked obstruction at the aortic orifice; indicates, therefore, the preponderance of retraction or crumpling, or it may be of ulceration over calcification of the valves, and this accounts for the amount of diastolic regurgitation revealed by the great collapse of the pulse, the distinctness of the diastolic arterial murmur, and to some extent also accounts for the distinctness of the diastolic murmur over the cardiac area. That, however,

depends partly on the condition of the remaining portion of the valves, and the degree in which they are capable of originating a fluid vein, though, no doubt, the force of the regurgitating current—which must be greater the more complete the regurgitation is—must have its influence in rendering this fluid vein more sonorous, while the condition of the sternum, and its connection with the ribs, has its influence in propagating the murmur over the chest. The points to which I wish specially to direct your attention at present are, the specialities of the aortic murmur, viz., the absence of a systolic murmur from the aortic area, and the presence of well-marked double murmur in the arteries. I have already stated that the absence of the systolic murmur from the aortic area is due to the almost entire absence of any obstruction at its orifice; some degree of obstruction there must be, however, and to that as well as to the same cause which makes the carotid murmur so distinct, it is due that in the innominate artery this murmur is more distinct than at the aortic area, though it falls very far short of that which is audible in the carotid artery, where the stethoscope, by compressing the artery, gives rise to a fluid vein which increases and intensifies the systolic portion of the murmur. The systolic arterial murmur, though intensified by compression of the artery, is quite audible without this, when we place the stethoscope in the immediate neighbourhood of the artery; because, though partly due to the formation of a fluid vein, it owes its existence much more to the fact that the greatly dilated and hypertrophous left ventricle sends forward a wave of blood disproportionate to the calibre of the arteries. Consequently we have in the carotid arteries, at the moment of the ventricular systole, a loud murmur composed partly of that produced by the fluid vein originating at the aortic orifice, but also, in a much greater measure, by the vibrations caused by the sudden tension produced in the arterial coats. We see, then, that a loud systolic murmur may be audible in the arteries in two very different conditions of the aortic orifice: where there is much

obstruction from calcified valves, the systolic murmur is loudest at the aortic orifice and is propagated into the arteries, gradually diminishing in intensity as we recede from the heart; where there is great destruction of the valves, and consequently but little obstruction, the systolic murmur is always less audible over the aortic orifice than over the arteries, and the diminution of the murmur on receding from the heart is not so great. In both cases the rhythm of the murmur alters as we approach the periphery, being always in unison with the rhythm of the pulse, and not with that of the heart, so that a ventricular-systolic murmur in the carotids may become ventricular-diastolic when it reaches the radial or femoral arteries; or if it still remain ventricular-systolic, then the cardiac systole is that of the pulsation immediately succeeding that which originates the murmur, as we may ascertain by tracing it down the arteries. The diastolic murmur, however, which in the carotids immediately follows the ventricular-systolic murmur, and which in every part of the arterial system is synchronous with the arterial systole, whatever relation that may have to the cardiac diastole, has but one origin, viz., free regurgitation into the ventricle. It is inaudible unless we compress the artery with the stethoscope so as to produce a fluid vein at that point; and there it is always to be heard with greater or less ease, according to the amount of regurgitation present. The readiness with which we hear this ventricular-diastolic murmur in the arteries may be accepted as a measure of the freedom of regurgitation into the ventricle, but its absence is by no means to be regarded as a proof that no regurgitation is present, as, where much obstruction exists, this murmur may be quite inaudible above the aortic valves. Thus, I agree with Jaccoud\* when he states that, on placing the stethoscope lightly over an artery at the moment of its diastole, only a slight membranous click is heard in the normal condition synchronous with its sudden expansion; a stronger pressure may increase this sound, but produces no distinct murmur.

\* *Leçons de Clinique Médicale*, Paris, 1869, p. 185.



When, however, the artery is forcibly dilated by the large blood-wave coming from a dilated hypertrophous ventricle, the very slightest pressure converts this membranous sound into a loud systolic murmur; should this be associated, as it usually is, with free regurgitation, a slightly increased pressure brings out clearly enough a soft diastolic murmur, succeeding the systolic one, while a still stronger pressure extinguishes all murmurs and ultimately all pulsation. I agree also with Duroziez\* in thinking this double arterial murmur to be in a great measure an artificially produced phenomenon—one certainly, the elements of which naturally exist in all suitable cases, but which can only be rendered audible artificially; but I differ entirely from him in his estimate of it as a phenomenon of importance in the diagnosis of aortic incompetence. In one respect I go further than Duroziez, for I have no hesitation in stating that a true ventricular-diastolic murmur audible in the arteries is never heard except when aortic incompetence exists;† but I have equally little hesitation in stating that aortic incompetence exists in many cases in which no such murmur is audible. Traube‡ believes that when aortic regurgitation is extreme, we can hear in the femoral arteries a double sound—not a double murmur—which originates spontaneously in the artery itself, without the intervention of the observer; the systolic sound arising from the vibrations produced in the arterial walls by their great and sudden distension, the diastolic sound being caused by the vibration produced by their equally sudden relaxation. But the idea of a diastolic sound due solely to *sudden relaxation* of the arterial coats is so untenable from a physical point of view, that there is little wonder that the element of truth upon which Traube's theory was based escaped, for a time,

\* "Du Double Souffle Crural comme signe de l'Insuffisance Aortique."—*Archives Général de Médecine*, April 1, 1861.

† Duroziez says that this double murmur is also heard in cases of enteric fever, chlorosis, and lead poisoning.

‡ *Gesammelte Beiträge zur Pathologie und Physiologie*, Berlin, 1871, Bd. i. p. 793; and *Berliner Klinische Wochenschrift*, 1872, No. 48, p. 573.

due recognition, more especially as the phenomena concerned have no particular relation to aortic disease, and are often well marked in widely diverse conditions. When we auscultate below Poupart's ligament, or at the root of the neck, or just below the clavicle, we can often hear one, two, or even three, short sharp sounds. In aortic regurgitation, one of these, always the second, if two sounds are present, or the third, if there be three, is the sound of arterial distention already referred to, and is readily transformed into a blowing murmur by pressure on the artery. The other sounds are most distinctly heard over the veins in the respective situations, and no amount of pressure ever converts them into murmurs, even when it can be readily applied, as over the femoral vein. Pressure may extinguish these sounds, but never changes them to murmurs. They are occasionally to be heard in cases of aortic regurgitation, at least as often in other forms of cardiac disease, or in pulmonary affections, and are always associated with tricuspid regurgitation. I agree with Friedreich \* in ascribing them to the sudden sharp closure by the regurgitant blood-wave of the venous valves in the situations referred to. Where the phenomena are produced in the veins alone, the sound may be single or double; in the first case it is produced by the ventricular systole alone, in the second the first sound is due to the auricular contraction, the second to that of the ventricle. These venous sounds may be heard in every form of disease of the heart or lungs capable of producing tricuspid regurgitation, but it is only when we have to do with free aortic regurgitation that the arterial element comes into play. The sound may then be double or triple, and the arterial portion is always the last; because of course the auricular contraction invariably comes before the systole of either ventricle, while the regurgitant wave from the right ventricle, favoured by the congested condition of the veins, precedes the pulse wave from the left ventricle, which is more than usually delayed

\* Ueber doppelton an der crural arterie, sowie über Tonbildung an den crural venen.—*Deutsches archiv für Klinische Medizin*, Bd. xxi. s. 205.



by the altered physical condition of the arteries (*vide* p. 87). Of course these sounds have not the significance which Traube ascribed to them, a double sound being not always present when regurgitation is extreme, and often to be heard when there is no aortic disease whatever; moreover, the venous sounds cease to be audible so soon as the congestion becomes great enough to make the venous valves incompetent.

In the normal condition of the heart and bloodvessels, it is generally supposed that cardiac pulsation is arrested at the capillaries, but this is by no means always the case; and even in those who believe themselves to be perfectly healthy, and whose condition certainly very closely approximates the normal, the cardiac pulsation is occasionally propagated not only into the capillaries, but also into the veins, as was first recorded by Quincke. Such cases are, however, really abnormal from failure of one or other of the three great factors of the circulation.\* These are (1) the force and frequency of the heart's action, which must suffice to keep the arteries overfilled, that is, to maintain the normal blood-pressure; (2) the peripheral resistance, that normal state of constriction of the arterioles called their tone, which keeps the arteries distended by preventing too rapid an outflow; and (3) the elasticity of the arterial coats, which when called into play by the two factors just mentioned, converts the intermittent supply from the heart into the continuous flow of the capillaries and veins. In aortic regurgitation the arteries are dilated, and their elasticity greatly lost, and though the blood-pressure is not low in proportion to the freedom of regurgitation, yet the conditions are always favourable for the production of a capillary pulse wave, which is more or less present, and tends to be more marked and to pass more distinctly into the veins the larger the ventricle and the more forcible its action. Hence a capillary or venous pulse—and in this case of course I speak of a direct venous pulse running up the hand and arm and not of jugular regurgitation—is frequently seen in cases of aortic disease,

\* Foster's *Physiology*, 3d edition, 1879, p. 136.

both in such parts as are in their natural condition, as in the cheeks, nails, and retina, as well as in inflamed or erysipelatous parts, and though by no means pathognomonic of regurgitation, it is yet an important indication of impairment of the circulation.\* In the spring of 1875 Bailey was again for several weeks under observations in Ward V. The cardiac phenomena continued unchanged, and he has again been discharged relieved.†

The next case is the last one I shall comment upon at present, and I bring it before you now, simply as an illustration of a third form in which the murmurs of aortic incompetence may occur.

CASE IV.—Matthew Murray, a police-officer, aged 39, admitted to bed 7, Ward V., on 19 March 1874, complaining of pain in the chest and breathlessness, from which he has suffered greatly since last October. He was originally a farm-servant, but has been in the police force for eleven years and a half. He is 5 feet 9 inches in height, stout and healthy-looking; has always been sober, well fed, clothed, and housed, and has never suffered from any serious disease—in particular, has never had rheumatism; seven years ago had sores on his penis, but they were not followed by any sore throat or eruption, &c. In his earlier days he was in the habit of making violent and laborious exertions; latterly, he has not. Has never been in the army, and cross belts on the chest form no part of his official costume. About seven years ago he received a severe blow on the chest from one of the shafts of

\* Vide Mr T. William King, *Guy's Hospital Reports*, Nos. iv. and xii.; Stokes on *Diseases of the Heart and Aorta*, Dublin, 1854, p. 202; Lebert, *Handbuch der Practischen Medicin*, Tübingen, 1862, vol. i. p. 725; Quincke, "Beobachtungen über Capillär- und Venenpuls," *Berliner Klinische Wochenschrift*, 1868, No. 34, p. 357; Grandclement, "De la Valeur des Battements de l'Artère Centrale de la Retine, dans les Affections Cardiaques," *Lyon Medical*, 1874, No. 12, p. 136.

† Bailey returned once or twice subsequently, and then ceased to appear; it is an interesting circumstance in connection with his case that to the last he maintained himself as a coal porter, and was daily in the habit of carrying bags of coal on his back to the upper stories of some of our highest houses.

a lorry while trying to stop a runaway horse. On inspection, marked pulsation is visible, both in the carotid and brachial arteries; the cardiac pulsation is defective, apex beat not noticeable, slight pulsation in the scrobiculus cordis; both pupils are normal; on palpation, the radial pulses are found both equal, jerking with a well-marked thrill, and beating 75 per minute. The thrill is not so perceptible in the carotid as in the brachial and radial arteries. The jerking character of the pulse is markedly increased on elevating the arm above the patient's head as he sits. The cardiac apex is scarcely to be felt; it beats between the fifth and sixth ribs, three inches and a half from the left edge of the sternum. The cardiac pulsation is generally defective. On percussion in the parasternal line, the dulness commences at the upper edge of the third rib, and extends down to the liver dulness. Transversely in the line of the fourth rib, dulness commences half an inch from the right edge of the sternum, and extends across for a distance of six inches. On auscultation in the mitral area, the first sound is heard obscured by a systolic murmur, which, on being traced up to the aortic area, is found to have its position of maximum intensity there. This murmur is audible over all the cardiac area, obscuring the first sound in the mitral, tricuspid, and pulmonary areas, and replacing it completely in the aortic area. In the pulmonary area an accentuated second sound is faintly heard greatly obscured by a diastolic murmur, which replaces the second sound in all the other areas, and is loudest in the aortic area. This double murmur is more or less audible over all the chest, both before and behind, but has its position of maximum intensity in the aortic area. The aortic dulness reaches to within one inch and a quarter of the top of the sternum. A distinct double murmur is heard in the carotid and femoral arteries. Dyspnœa and cough are chiefly remarkable when the patient lies down at night. Pulmonary percussion normal over the left lung, slightly higher in pitch (emptier) over the right lung, especially over its lower lobe. Pulmonary auscultation normal over the left

lung; over the right lung the vesicular murmur is weakened, especially over the lower lobe. In other respects the patient is normal.

After what has been already said, this case requires little explanation. The extent of the transverse dulness might lead to the supposition of considerable dilatation of the right side, but the trifling character of the impulse in the *scrobiculus cordis* is against this idea. Again, the very free regurgitation through the aortic valves is opposed to the idea of the impulse between the fifth and sixth ribs being that of the true apex; while the very considerable force of the blood-wave, as perceived in the arteries, is also opposed to the idea of the defective ventricular impulse being a true measure of the condition of the cardiac walls. We are therefore forced to the conclusion that the great breadth of the cardiac dulness is due to the oblique position of the heart, which lies more transversely and also deeper than usual, the defective impulse being due more to the increased depth of lung covering it than to any preponderance of dilatation over hypertrophy; and the cause of this pushing of the heart backwards and to the left, is to be found in the condition of the aorta, which is largely dilated, as proved by its increased breadth of dulness, and has an aneurismal bulging on its ascending portion just above the heart, pressing upon the right bronchi, and especially upon that distributed to the lower lobe of the right lung, as revealed by the alteration in the percussion note over that part of the lung, and also by the enfeebled and defective respiratory murmur audible over the corresponding part of the chest. The aorta is probably in this case in the state of a cirroid aneurism, largely dilated, with at least one bulging just above its commencement. At present, however, we have to deal with this case simply as an example of a third mode in which the murmurs of aortic incompetence are produced and propagated. This patient has never suffered from rheumatism, and the incompetence of his aortic valves is probably entirely due to the dilatation of his aorta. As I have already shown, and shall by and by illustrate,



the first effect of aortic dilatation is the forcible closure of the aortic valves, and the production of a markedly accentuated aortic second sound.\* As this dilatation proceeds, the valves become too narrow perfectly to close the large orifice, and accompanying this accentuated second sound we have a diastolic murmur of greater or less intensity. Though such an aorta must be atheromatous, there may yet be no diastolic murmur audible above the valves; if, however, it be roughened by projecting spiculæ, the diastolic murmur may commence high above the valves, and in any case it is frequently so loud as completely to obscure the aortic sound. In the earlier stages of this affection, appropriate treatment not infrequently so favours the contraction of the vessel as to cause the complete vanishing of the diastolic murmur, the second sound remaining, however, permanently accentuated, while the diastolic murmur is in such circumstances, as may be supposed, exceedingly apt to recur. In all such cases the valves are seldom much calcified, frequently they are quite flexible, and when they are so, they present no obstacle to the egress of the blood, the systolic portion of the murmur being sometimes, though probably rarely, due to the production of fluid veins by projecting spiculæ, more frequently to the tensile vibrations produced in the arterial walls by the wave of blood disproportionate to their calibre, sent forward by the dilated and hypertrophous ventricle, but by far its most common and most important source is the production of fluid veins in the blood passing through the comparatively narrow orifice into the aneurismal dilatation beyond. If there be spiculæ projecting from the interior of the aorta, the diastolic murmur may be partly due to their influence; it is always, however, mainly due to the production of fluid veins at the aortic orifice, which forms a comparatively narrow strait between two parts of wider calibre—the dilated aorta and the ventricular cavity. This diastolic murmur has its position of maximum intensity at or below the aortic orifice, and owes its propagation over the cardiac area to the resonant

\* *Vide antea*, p. 31.

qualities of the sternum and ribs. The double character of the arterial murmur is due to causes precisely similar to those which have been shown to have the same effect in the immediately preceding case, and has exactly the same significance.

From the foregoing cases we learn, that amongst all the various signs which enable us to detect aortic incompetence or predict its significance, the murmurs which accompany it, and their mode of propagation, are not the least important, and we must always remember that though a diastolic murmur at or beneath the level of the aortic valves, in the line of the sternum, is pathognomonic of aortic regurgitation, yet in regard to prognosis, the ascertaining of the mode of propagation of this murmur is of much more consequence than its mere detection.

The important points which have just been illustrated may be summarised as follows:—A diastolic murmur at or below the level of the aortic valves, chiefly audible in the line of the sternum, is significant of aortic incompetence. If this diastolic murmur be inaudible in the carotid arteries, it is invariably accompanied by a systolic murmur, having its maximum intensity at the aortic valves or in the so-called aortic area, and this indicates comparatively trifling incompetence with considerable obstruction at the aortic orifice, most probably from calcified semilunar valves. If this diastolic murmur be distinctly audible in the carotid arteries, it is invariably preceded by a loud systolic murmur in them, the systolic portion of the murmur being not always very audible in the aortic or in any part of the cardiac area, and this indicates very considerable incompetence with comparatively trifling obstruction. What has been stated as to the murmurs in the carotid arteries refers equally to those in the femoral as well as in all other arteries. Of course, each case differs from another, and as in practice the most marked examples of great regurgitation with slight obstruction are found passing into instances of great obstruction with slight incompetence, so the murmurs indicative of these lesions are practically found to vary indefinitely. The cases which have been selected for illustration

are, however, tolerably well-marked and pure examples of the three principal forms in which the murmurs due to aortic incompetence present themselves. A double murmur in the arteries being always the result of great incompetence, and if associated with a history of rheumatism, due to excessive retraction and possibly ulceration of the semilunar valves, and accompanied by an absence, more or less well-marked, of the systolic murmur from the cardiac area ; while, if this phenomenon be not associated with a distinct history of rheumatism, the systolic portion of the murmur will be found to be well marked over all the cardiac area, and the lesion will be arterial (aortic dilatation) and not primarily valvular in its origin.

## LECTURE III.

ON INCOMPETENCE OF THE AORTIC VALVES, WITH SPECIAL REFERENCE TO THE PROGRESS OF THE DISEASE, ITS PROGNOSIS, AND TREATMENT.

GENTLEMEN,—In the previous lecture on Aortic Incompetence I have directed your attention specially to the diagnostic significance of the murmurs accompanying it, because it is from them we learn the comparative freedom of the regurgitation present, upon which much of the prognosis and many of the consecutive phenomena depend.

When regurgitation takes place through the aortic valves, the left ventricle, during its diastole, fills from two sources instead of one—from the aorta as well as from the auricle. The first and most immediate effect of this is a sudden diminution of the arterial tension, which impresses its character on the pulse in exact proportion to the amount of regurgitation present—a fact which shall be immediately more particularly referred to. The second effect of this diastolic filling of the ventricle from two sources is, that when the systole of the auricle takes place there is produced, first, a greater or less over-distention of the left ventricle ; and, second, an incomplete emptying of the left auricle. The result of the latter is a greater or less amount of passive congestion of the lungs, revealed—as I have already pointed out, every obstruction to the onward flow of the blood through the heart, that is, every passive congestion of the lungs from a cardiac cause,



is revealed—by an accentuated pulmonary second sound. Once started, this passive congestion of the lungs must, of course—if no compensating change were possible—go on increasing with each cardiac pulsation, until at length it affected the right ventricle, over-distended it, opened up the tricuspid valve, and ended by producing passive congestion of the general systemic veins, terminating in general dropsy, and a gradual lowering of the arterial blood-pressure till it became reduced below that compatible with life, and death ensued under symptoms of a gradually increasing asthenia.\* This is, in fact, a very common mode of death in other forms of cardiac disease; but it is a comparatively rare one in aortic incompetence, because in it the lesion which obstructs the onward flow is of such a character as to be capable of producing, directly and at once, such a fall in the arterial blood-pressure as is only brought about by other lesions indirectly, and after a long lapse of time.

In all cardiac lesions, however, there is a tendency to postpone this ultimate result for a longer or shorter time by the production of secondary changes in the heart itself, which more or less perfectly compensate these lesions; and it is the object of treatment so to further or to regulate these changes as to procure as perfect a compensation as is practicable, and to maintain its efficiency as long as possible.

In aortic incompetence, as it is ordinarily seen in those who come to consult us, the overloaded condition of the heart gives rise to various distressing sensations, revealed by uneasiness or pain in the cardiac region, with irritability of the organ, evinced by palpitation on the slightest provocation, direct or reflex, as the most trifling exertion, or the mere ingestion of

\* “Systemic death consists in decline of aortic pressure. This decline may occur rapidly, as in syncope; but usually, even in deaths by violence, it is very gradual. In deaths from disease it may last (gradually decreasing) for days, weeks, or even months.”—*Handbook for the Physiological Laboratory*, p. 209.

food. This, however, is not the normal condition of such sufferers, it is a state of disease, a state of ruptured compensation, which it must be our endeavour to remedy. When aortic incompetence is established, and the ventricle fills from two sources instead of one, the first effect is of course to overfill the ventricle, and the earliest attempt at compensation is the accommodation of the left ventricle to the increased supply of blood it is required to contain. This is readily brought about by the elasticity of the muscular structure of which it is composed. The first step towards compensation is therefore dilatation of the left ventricle. Inasmuch, however, as simple dilatation of the ventricle can only provide a receptacle for the blood delayed, and not a means of restoring it to the circulation, this first step would postpone the inevitable end only for a very short period, and must itself be compensated before the balance of the circulation can be so efficiently restored as to render the lesion mute. To this end the dilated ventricle hypertrophies; *pari passu* with the dilatation of its cavity, its walls become thicker and more powerful. The result is, that as the left ventricle dilates it also hypertrophies, and that in cases of disease in which free regurgitation is only slowly developed; this all takes place with so little disturbance of the cardiac or of the general circulation, that it is often only after the lapse of many years—frequently passed in strenuous labour—that the patient learns to his astonishment that he has in all probability been for so long the subject of heart complaint.\* On the other hand, when regurgitation has been suddenly developed in an extreme degree, as in cases of accidental rupture of one or more of the segments of the aortic valves, the immediate effect has occasionally been great disturbance of the cardiac circulation, and complete compensa-

\* I found this statement on the fact that I have seen many cases of aortic incompetence in whom the compensation had been accidentally or unavoidably ruptured twenty years or more subsequent to one single attack of rheumatic fever, from which, in all probability, the lesion dated, coupled with my own knowledge that some have suffered for quite half that time from aortic regurgitation without being aware of it.

tion has never been perfectly developed, the patient continuing to suffer till in no long time death has ensued.\*

When aortic incompetence has been developed as the result of local disease in a moderately healthy subject, in whom dilatation and hypertrophy of the left ventricle have gone on, *pari passu*, in such a way as to develop and maintain what is called a perfect compensation for the existing lesion, a time at length arrives when this is no longer possible. I may be unconsciously doing injustice to those who have already written on this subject, but it seems to me that they have assumed that when this so-called perfect compensation is once attained,—it may be after a longer or shorter period of cardiac perturbation,—it is maintained, accidents excepted, till nutritional changes in the heart walls render its longer continuance impossible.† If this view, however, be correct, too much attention has been bestowed upon what we may call the vital forces, and too little upon the simple mechanical agencies involved in aortic regurgitation—agencies which are, however, of vast importance in producing injury, and which must be

\* Balthazar Foster, in his *Clinical Medicine*, p. 139, says, that four years and a half covers the date of death in all such cases of traumatic lesion of the aortic valves. There are many clinical facts to prove that this is by no means always the case. I have seen cases in which an aortic cusp has been torn from its attachments, the lesion healed, and in whom serious symptoms were not observed till years subsequently. Experimental investigation, as we shall presently see, is entirely at one with this experience, and opposed to the idea of such an accident being of necessity and always speedily fatal.

† Thus Jaccoud says—"Quelque parfaite que soit la compensation des lésions cardiaques, elle est temporaire ; l'équilibre artificiel au moyen duquel elle maintient une circulation à peu près normale peut durer des années, mais il faut qu'il se rompe, cela est fatal ; le tissu musculaire du cœur atteint dans sa nutrition subit à la longue une transformation régressive, généralement grasseuse, et lorsque cette altération secondaire, dont Paget et Stokes ont montré toute l'importance, présente une certaine étendue, le ventricule, quoique augmenté de volume, quoique hypertrophié en apparence, ne contient plus assez d'éléments contractiles pour faire face au travail excessif que la lésion lui impose ; sa force propulsive diminue, il se vide mal, l'évacuation du système veineux est par suite gênée, la compensation est détruite, et comme cette rupture résulte d'une condition anatomique sur laquelle nous n'avons aucune prise, il faut que le cœur s'arrête, et que le malade succombe."—*Leçons de Clinique Médicale*, Paris, 1869, p. 215.

duly attended to and counteracted in our therapeutical attempts to alleviate the mischief produced by them. Thus, when a leakage, however trifling, has once been established through the segments of the aortic valve, the effect of this—with the patient in the erect position—is, according to Pascal's law,\* that the interior of the left ventricle is being constantly dilated by a force equivalent to the weight of a column of blood the height of the cranium above the heart, and of the diameter of the ventricular lumen. From the moment that leakage is once established till the cessation of life, this hydrostatic pressure never ceases, though it may be modified by position; its action is not only continuous, but it increases, in accordance with the law referred to, *pari passu* with the gradual dilatation of the ventricle;† while all that the organism has to oppose to this powerful dilating force is the practical exercise of that well-known but inexplicable law, whereby all hollow involuntary muscles hypertrophy and increase in strength in proportion to the amount of any obstacle preventing the exercise of their function (Paget). A due consideration of what has just been said will sufficiently explain the danger to which such patients are subjected, as well as the manner in which nutritional derangements, either general or local, may increase that danger. For instance, if, at the moment that aortic leakage is established, the system is in a depressed condition of nutrition, there may be a difficulty, or even an impossibility, of establishing compensatory hypertrophy, and the result may either be a more or less prolonged period of cardiac disturbance, or sudden death from asystole. And similar results may arise from a similar cause at any moment—even long after compensation has been established—during the

\* Pressure exerted anywhere upon a mass of liquid is transmitted undiminished in all directions, and acts with the same force on all equal surfaces, and in a direction at right angles to their surfaces.—*Vide Traité de l'Équilibre des Liquéurs*, &c., par Blaise Pascal, Paris, 1763.

† In striking contrast to this view, it is somewhat singular that Rindfleisch says of this same lesion—"This dilatation is a result, not of the insufficiency, but of the stenosis."—*Vide Manual of Pathological Histology*, New Sydenham Society's Translation, vol. i. p. 291.



persistence of the leakage; that is, at any after-period of the patient's existence. It is, however, in the history of this lesion subsequent to the establishment of compensatory hypertrophy that a great part of its interest lies, especially in regard to the influence of local nutrition. If we regard the compensation, once established, as perfect and complete, then it is obvious that, apart from accidental disturbances, we must wait the development of local nutritional changes before we can have any disturbance of the compensation. If, however, we look upon the compensation as always incomplete, though practically sufficient for the carrying on of the circulation, then a time must at length arrive when the compensation is ruptured from purely physical causes, and that quite independent of either general or local disturbances of nutrition; and it seems to me that this is the most usual case.

In aortic regurgitation there are three possible conditions of the left ventricle, for it is to the state of that cavity that our inquiry in this matter is practically limited.

1st, We may have a condition of over-compensation, in which the left ventricle is hypertrophied in excess of its requirements, and which is said to give rise to many disagreeable and possibly dangerous symptoms. This is a state of matters which is constantly referred to by all recent writers on the subject. It must, however, be one of extreme rarity, if it occur at all. In my own experience I can only recall one case of aortic regurgitation in which I ever had even a suspicion of such a state of matters, and that occurred so long ago that I am by no means certain that, were a precisely similar case to come before me now, I would acknowledge it as a definite example of over-compensation. At all events, I know not where to find recorded any well-authenticated case of the kind; and I am persuaded that for all practical purposes we can well afford to discard all consideration of such a condition.

2d, The balance of compensation may be perfect; the amount of hypertrophy may be just sufficient to counterbalance the

dilating effect of the regurgitation, and with a slightly dilated and hypertrophied heart the circulation may go on as before, till, from accidental or inevitable nutritional changes, the compensation is ruptured, and disturbance of the circulation sets in.

3d, We may have, from the moment that aortic leakage sets in, a dilating power acting on the left ventricle, which from physical causes, must gradually increase. This dilating force acts as a hindrance to the onward current of the blood; the muscularity of the left ventricle reacts against this obstacle, and for a time successfully, so far as the maintaining of the circulation is concerned, but always at a disadvantage. The dilatation which commenced the organic disturbance continues ever in advance, the compensating hypertrophy which followed continues ever to lag behind, till at length a period is reached when nutritional disturbances are produced by structural causes, and asystole is threatened, because the weight of the dilating fluid is in excess of the contractile force of the ventricle. At this moment it is fortunate for the patient that the increased weight of the blood is, from physical causes, always accompanied by increase of dilating power, so that just at the moment that ventricular paralysis is threatened, a way of escape is prepared, the segments of the mitral valve give way, and, by regurgitation into the left auricle, the overburdened ventricle is relieved and the inevitable end postponed. Every one must recognise in this latter sequence of events the ordinary history of a case of aortic regurgitation, and must also be fully aware, that even after the occurrence referred to, a patient may die with his ventricular muscle in a state of comparative health. We must all, therefore, be fully prepared to acknowledge the paramount importance of Pascal's law in the sequential production of the organic changes consequent on aortic regurgitation, quite apart from nutritional changes.

But though the hydrostatic influence developed by the imperfect condition of the aortic valve inevitably gives rise to

the conditions referred to, these may be precipitated by any general or local disturbance of nutrition. Whenever from general debility the hypertrophy of the left ventricle is interfered with, asystole and sudden death may occur at a very early period of the affection, or over-dilatation and mitral regurgitation may take place at a similarly unusually early period. This may be afterwards compensated, but is probably always followed by an exceptionally early fatal termination. Similar results are threatened at any period of the disease when general nutrition is accidentally interfered with by privation, overwork, or any febrile condition, so slight even as an ordinary catarrh. It is under these circumstances that the disease, if it have ever been mute, ceases any longer to be so; cardiac disturbance sets in, and the patient comes under treatment. In these cases the ruptured compensation may still be restored by appropriate management, and the patient may be reinstated in comparative health. It is otherwise, however, when the nutritional disturbance is local, and solely due to the progress of the disease. Judicious treatment may still prolong life, but efficient compensation can no longer be restored, and though the end may be postponed, it is inevitable. The importance of the heart as the central organ of circulation, and the amount of work which, as such, it is needful for it to perform, coupled with our knowledge of the paramount importance of food in relation to work done by organic frames and their constituent organs, enable us readily to understand how any defect in the nutritional qualities of the blood, however brought about—by old age, cachexia, or disease—may impair the working power of the heart. And when we know that this diminished contractile force is opposed to an obstacle which from physical laws must, if uncompensated, still further impair the physical means by which that force is applied, we can easily understand the evils to which a patient in this condition may be exposed.

It is impossible, however, to have any clear ideas as to the manner in which aortic disease progresses, its probable duration,

or its treatment, without a thorough knowledge of the physical agencies implicated in that progression, and the mode in which the heart is enabled to resist them. Following Stroem,\* Thibesius,† and Brücke,‡ I formerly supposed that the heart was flushed with arterial blood only during the diastole, for these reasons:—1st, because the blood-current flows at right angles to the orifices of the coronary arteries, so that only a small portion of the systolic wave could be supposed to enter them; 2d, because the ventricular tissue is so firmly contracted as still further to limit the systolic supply; and 3d, because in a considerable number of individuals the coronary arteries open so far within the valvular zone, that at the moment of ventricular systole their orifices are closed by the segments of the aortic valve thrown back on the wall of the artery by the advancing blood-wave, so as to shut off completely the possibility of any systolic flushing. These reasons, if correct, are sufficient to justify the postulate started with. But Gaskell§ has shown that even tetanic muscular contraction favours the arterial blood-flow through it, instead of obstructing it. Hyrtl|| has shown, from a special examination of 117 bodies, that in the greater number the orifices of both coronaries are above the free margin of the semilunar valve, and that one at least always is so. He also states, as the result of numerous experiments on rabbits, cats, and dogs, that section of the coronary arteries is invariably followed by an intermittent blood-spurt coincident with the systole of the ventricle, and this he thinks conclusively proves that closure of the coronaries by the segments of the aortic valve cannot

\* Haller, *Elem. Physiol.*, vol. i. lib. iv. s. v. §§ 18, 19, Lausannæ, 1757.

† *Dissertatio Med. inaug. de circulatione sanguinis in corde*, Lugd. Batav. 1708, §§ 24, 25.

‡ *Physiologische Bemerkungen ueber die Arteriæ Coronariæ Cordis*, Sitzb. d. K. K., Acad. der Wiss. zu Wien, Cl. Bd. xiv., 1854, s. 345.

§ Ludwig's *Arbeiten*, 1876; *Journal Anat. and Phys.*, xi. 360.

|| *Beweis dass die Ursprünge der Coronar-Arterien während der Systole der Kammer von den Semilunar Klappen nicht bedeckt werden.*, u.s.w., Sitzb. d. K. K., Akad. d. Wiss. Cl. Bd. xiv. s. 373.



exist.\* By a reference to the physics of the valve segments themselves, and also of the blood-current, Ceradini has shown that the valve segments are not closely applied to the arterial wall during the ventricular systole, but float in an intermediate position, maintained in equilibrium by the central or axial stream on the one hand, and on the other by reflux currents which, originating at the exterior of the axial stream, flow outwards and downwards against the arterial wall, and are reflected from it upon the posterior surface of the segments of the aortic valve.† These statements seem conclusively to prove that the coronary arteries are patent to the blood-flow during the ventricular systole; and when we reflect that the coronaries are flushed not merely by a reflux current of unknown value, but, through the influence of Pascal's law, by a blood-current having the minimum aortic pressure of 200 mm. Hg., we see that the cardiac muscle is freely supplied with highly oxygenated and nutritive blood at the very moment when it requires it most, when the transformation of energy, from potential to kinetic, within its substance, is at its height. And this is in complete consonance with the fact that, except in very advanced cases of long standing, or where other causes have also been at work, the heart in aortic incompetence is always found to be well-nourished and free from signs of degeneration. It also explains the remarkable fact that sufferers from aortic disease are not necessarily shortlived

\* *Ueber die Selbststeuerung des Herzens, ein Beitrag zur Mechanik der Aorta Klappen*, Wien, 1855, s. 59, ff. The self-regulative power of the heart, that is the supposition that the flushing of the coronaries is necessary for the diastolic dilatation of the heart, is shown to be a myth by the facts that the heart of a frog or of a shark beats for hours although empty of blood, and also by the experiments of Panum and Von Bezold, who found that the hearts of dogs and rabbits beat for hours after complete blocking of the coronary arteries. Vide *Virchow's Archiv*, Bd. xxv., 1862, s. 308, &c.; and *Untersuchungen aus dem physiologischen Laboratorium in Würzburg*, Erster Theil, 1867, s. 256, &c.

† *Der Mechanismus der halbmondförmigen Herzklappen*, Leipzig, 1872. Ceradini's work contains a very complete epitome of the literature of the subject.

from causes which shall be presently explained, they are certainly liable to sudden death in a higher ratio than other men, but they often lead useful and wonderfully long lives, quite unconscious of the existence of any serious disease. To others the signs, or some of them at least, may be apparent enough; they themselves are quite unconscious of anything but the fullest vital vigour. One of the most remarkable instances of this which I have ever seen, was that of a clergyman who had his first and only attack of acute rheumatism thirty-four years before he consulted me. His disease when I saw him first was evidently far advanced, and only too surely approaching its termination, yet he assured me that till two or three months previously he had been perfectly unaware that anything ailed him. He was a married man with a family, and knew that all his life his heart had beat stronger than other hearts did, but he rather regarded this as a sign of vigour than the reverse. Over exertion at lawn tennis precipitated the break down of his compensation, which was too near its natural close to permit of recovery, and he died three months after I first saw him. I have seen many similar cases, but none in which the history was so distinct, or the period between beginning and end quite so long, yet he was a man who discharged faithfully all the duties of his station, and took his share of such minor athletics as lawn tennis, curling, &c. Indeed, such patients are largely exempt from the troubles that afflict those labouring under mitral disease; the frequent attacks of bronchitis, of dropsy to a greater or less extent, or the multi-form uneasiness entailed by cardiac weakness, from which mitral patients suffer so much and so often, are all unknown to those labouring under aortic incompetence. It is well it is so, for such accidents are of much more serious import in the latter class of cases than in the former. The special immunity from cardiac symptoms enjoyed by those labouring under well-compensated aortic incompetence has been also noted by Niemeyer, who says that "such persons are frequently not even short of breath—a symptom never missed in cases of

mitral disease.”\* And he makes special mention of a huntsman from Greifswald, who, though labouring under extensive stenosis and incompetence of the aortic valve, with immense eccentric hypertrophy of the left ventricle, was yet able to go through all the manœuvres and forced marches of the Franco-Prussian war without difficulty ;† and there is no difficulty in either understanding or believing this remarkable immunity if we consider the manner in which the heart is nourished, and the mode in which compensation is established. Now and then it happens that a morbid anatomist holds up a heart and allows us to see water trickling through an aortic valve, the incompetence of which had not been detected during life. There is no need to be ashamed of this, for a certain force of stream is requisite to produce a fluid vein, and without a fluid vein there can be no murmur to aid us in making our diagnosis. But the crumpling of a rheumatic valve goes on so slowly that I myself see no reason to doubt that leakage has been long established before it attain force enough to reveal itself by a murmur. During all this time that the leakage has been imperceptibly advancing to its full development, dilatation of the left ventricle with compensatory hypertrophy have been equally gradually brought about through the agency of the physical and pathological laws formerly referred to,‡ so that we never hear a murmur of aortic incompetence without, at the same time, being able to detect some degree of eccentric hypertrophy of the left ventricle. Where the disease is arterial in its origin, and depends primarily on aortic dilatation, accentuation of the aortic second often long precedes any other sign, and it may even be associated with a systolic murmur of aortic origin for an indefinite period before the development of the signs of aortic patency,

\* *A Text-Book of Practical Medicine*, London, 1871, p. 347. This immunity of aortic patients from pulmonary symptoms is also referred to by Rosenstein, *vide* Ziemssen's *Cyclopedia of the Practice of Medicine*, vol. vi. p. 139, London, 1876.

† *Loc. cit.*

‡ *Vide antea*, p. 76.



as has been pointed out by Stokes, who distinctly recognised these signs as early indications of a progressively advancing incompetence,\* which probably begins as slowly and as imperceptibly as in the rheumatic heart. Even where incompetence is suddenly developed by rupture of one of the aortic cusps, there is no reason to suppose that the accident is always attended by serious disturbance of the circulation. At least, I have occasionally observed cicatrices at the root of depressed and incompetent cusps, where there was no history of any sudden disturbance, and where circulatory troubles have only arisen after an indefinite period of calm following an accident, to which the rupture of the cusp seemed to be due. It is true this is not always the case, and in some these troubles arise quickly and rapidly progress to their close, but the reason for this would seem rather to depend on the preceding state of the cardiac muscle than on the nature or position of the lesion. Rosenbach† has shown that when the aortic valves of healthy animals are suddenly destroyed there is no intermediate period, however short, of cardiac disturbance, but that the heart at once adapts itself to its altered condition, without a trace of any fall in the arterial blood-pressure, such as must have been observed had there been but a second's falter on the part of the heart. The dilatation‡ necessitated by the overfilling of the left ventricle into which blood flows from two sources instead of one, is provided for by the natural elasticity of the ventricular walls, and the increased power needed to expel the surplus blood is readily supplied by the reserve force of the heart, an organ which in health has always a considerable margin of latent energy upon which the maintenance of life itself depends in many morbid conditions. Under the

\* *Diseases of the Heart and Aorta*, Dublin, 1854, p. 227.

† "Ueber artificieller Herzklappenfehler," von Dr Ottomar Rosenbach.—*Archiv für experimentelle Pathologie und Pharmacologie*, Leipzig, 1878, s. 1.

‡ Rosenbach has fallen into the mistake of supposing that this dilatation differs in character from that occurring in other circumstances, stating that while in this disease dilatation precedes hypertrophy, in other forms hypertrophy precedes dilatation, *op. cit.*, s. 12. The only differences are those of degree, the mode of production is essentially the same in all cases.



influence of Paget's law the ventricular muscle slowly hypertrophies, and the reserve energy is gradually supplemented and ultimately replaced by an actual increase of muscular force. In this way dynamic compensation is for long efficiently maintained, but perfect structural compensation is never attained, the hypertrophy always lags behind the dilatation. By adopting Ceradini's view of the mode in which the heart is nourished, we can readily understand how this imperfect structural compensation may long preserve its dynamic perfection, ever slowly advancing towards its natural termination when the hypertrophy has outgrown the feeding powers of the coronary arteries. Then the heart ceases to grow stronger, and if its walls still grow thicker this is due to venous congestion and the development of fibrous tissue; dilatation rapidly advances, by and by the segments of the mitral valve cease to meet, mitral regurgitation is established, and if the patient is spared so long, though still liable to death from sudden asystole, the natural termination of his disease is now gradual asthenia and dropsy. This is the normal history of aortic incompetence, and it may embrace a period of not less than thirty-five years, though from the numberless accidents which may rupture the dynamic compensation, it is seldom permitted to last so long. Rosenbach has experimentally demonstrated the truth of the mode in which aortic disease progresses, which I believe I was the first to suggest,\* but Ceradini's explanation of the manner in which the heart is nourished is even more important, as it affords an intelligible explanation of the way in which the heart is enabled to carry on its function, apparently unimpaired, in spite of the existence of serious valvular lesion. The history of aortic regurgitation is well fitted to awaken in our minds the highest respect for the recuperative power of the heart, even when irremediably injured. It also teaches us the unadvisability, to say the least of it, of treating a disease of this character with only signs and no symptoms, clearly

\* *Ed. Med. Journal*, Feb. 1875, p. 709, &c. See also 1st edition of this work, p. 74.

showing that in these cases our duty is to watch and wait, carefully avoiding the *nimia diligentia*, yet prepared at any moment to act with energy and firmness.

The pulse of simple aortic regurgitation is, as was first described by Sir Dominic Corrigan, something entirely *sui generis*. In its most typical form it is large and full, quickly projected against the finger, and falling off rapidly. Sometimes it communicates a vibratile jar to the fingers. Scarcely has the shock of the impulse been perceived than the arterial tension sinks to a minimum, from the aortic reflux, and the sensation vanishes. This pulse—*pulsus celer et infrequens*—has been likened to the successive propulsion of shot against the finger, or to the jarring shock of the toy called a water-hammer. It is always regular, and its characteristics are increased by raising the extremity (arm or leg) in which we feel it perpendicular to the body as it lies horizontal, or by raising the arm above the head in the erect posture. But these characteristic peculiarities of this form of pulse vary with each case, and may be greatly modified. Thus great obstruction, which also diminishes regurgitation, may lessen the distinctness of the jar very considerably, while a certain amount of anæmia often increases it; and consecutive or coexisting mitral disease frequently impresses upon it the mitral characters of smallness, feebleness, and irregularity so strongly, that its own peculiarities are entirely lost. Occasionally in these circumstances elevation of the arm brings out with tolerable distinctness the collapse of aortic regurgitation; but this is not always easily detected, and cannot be relied upon unless well marked. When it is distinctly recognisable we are, however, justified by the twofold nature of the pulse character in assuming the existence of a double lesion—aortic and mitral.

If we feel the apex beat of the heart and either of the radial pulses simultaneously in a perfectly healthy individual, it will be found that they do not beat simultaneously, but that the radial pulsation lags behind that of the heart by a distinctly appreciable interval of time, which may vary slightly in each indi-

vidual, with special relation to the rapidity of the heart's action ; but the radial pulse in health always precedes the second sound. This delay of the pulse is due to the fact that the arteries are elastic and not rigid, and that the blood moves along them as a wave, and not merely as a mass, one end of which is extruded as the other intrudes.\* So long as the arteries are fairly distended, trifling variations exert no marked influence on the state of the arteries, or on the rapidity of the blood-wave ; and the same is the case in regard to the force of the heart's contraction. When, however, the heart's impulse is greatly increased, and especially if the blood-wave is enlarged, then the elastic arteries both lengthen and dilate. As in aortic regurgitation, the left ventricle is both hypertrophied and dilated ; the impulse is greater and the blood-wave larger than in health. The arteries are therefore longer (more tortuous) and more dilated than in health, so that the pulse is more visible, and attended by more arterial movement than usual (locomotive pulse). But as these phenomena may be due, though usually in a less degree, to other causes than aortic regurgitation, they are not distinctive of it, though their presence certainly leads us to suspect its possible existence. In aortic regurgitation, however, the arteries are not only longer and more dilated than usual, but, from the reflux into the ventricle, they are also less distended ; the blood-wave is therefore longer of reaching its maximum, and the pulse is delayed.†

\* It is extremely interesting to find Erasistratus as accurate in this matter as he is in his pathology, and Galen as wrong as he is—as usual—dogmatic. We are all the more safe in making these statements that it is from Galen's writings alone that we learn his great opponent's views : “ Nam quod ait Erasistratus, semper priorem (scil. arteriæ partem) moveri prius videri, sensus non probat.” —*Galen de Puls. Different.*, lib. i. c. 25. And again : “ Quod Erasistratus mentitur de rebus evidentibus. Omnes enim clare cernunt, omnes partes arteriarum eodem distendi tempore.” —*Galen de Causis Puls.*, lib. ii. c. 8.

† The pulse-wave seems to pass through the arteries like a tidal bore wave, as was first pointed out by Mr Colt.—*London Med. Gazette*, vol. xxxvi. p. 456. Practical experiments in proof of this are as yet wanting ; but the modern idea (*vide* “ Handbook for the Physiological Laboratory,” p. 228), that the arteries dilate and contract again in segments, to pass on the blood-wave, is open to the objection that the primary contraction of the aortic segment would necessarily

This delay of the pulse in aortic reflux is invariable, as was first pointed out by the late Dr Henderson more than forty years ago,\* the only apparent exceptions being those cases in which the delay is so great that the radial pulse coincides with the ventricular systole immediately succeeding that which has produced it; but any possible mistake in such a case may be corrected by tracing the pulse-wave from the ventricle through the carotid and brachial arteries to the radial artery, which may be fairly enough done by the hand, but may be quite accurately determined by the electric methods devised by Dr M'Kendrick† and by Dr Landois.‡

Whenever, therefore, we have a visible, locomotive pulse, which is delayed beyond the commencement of the relaxation

Fig. 1.

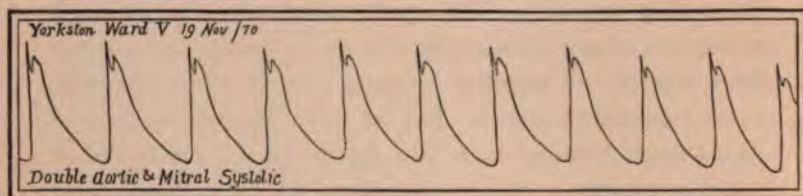
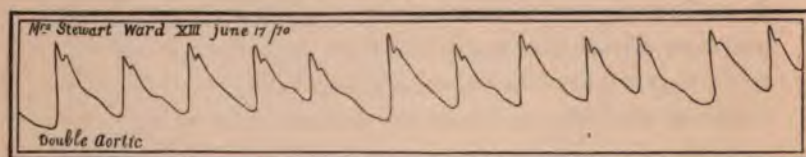


Fig. 2.



of that ventricular systole which has originated it—as marked by the occurrence of the pulmonary second sound—then we have to do with an aortic regurgitation which is probably great in proportion to the delay of the pulse. This form of pulse is represented graphically as in the two preceding figures. close the aortic valves long before the pulse-wave had reached the distal arteries, which we know is not the case.

\* *Ed. Med. and Surgical Journal*, vol. xlviii., 1837, p. 369.

† *Ed. Med. Journ.*, July 1874, p. 8.

‡ *Die Lehre vom arterien Puls*, von Dr Leonard Landois, Berlin, 1872, p. 302, &c.



In these tracings there is nothing to indicate undue postponement of the pulse. I have just pointed out how that is to be ascertained—here we have only noted the local succession of events as they are found to occur in any artery relaxed in all its dimensions, and more or less imperfectly filled. It is by no means necessary to produce such tracings as those shown that the aortic valves must be incompetent, it is sufficient that the arteries from any cause be relaxed and less full than usual; hence, in old age, when these conditions frequently exist in a considerable degree, the pulse tracing, even when the aortic valves are perfectly healthy, may be undistinguishable from that of aortic regurgitation.\* The pulse-wave consists of two parts—a percussion wave transmitted by the arterial walls, and a pressure or propulsion wave of blood which passes within the arterial lumen. In most pulse tracings the effect of both of these is conjoined in producing the abrupt line of ascent with which every pulse trace commences, and which is large and pronounced in proportion to the force of the beat and the size of the blood-wave; most developed, therefore, when the left ventricle is hypertrophied and dilated from any cause. When, however, there is delay of the pressure wave, as happens when unfilled arteries are longer than usual of reaching their maximum distention, then exactly in proportion to the difference of the rate of propagation of the percussion and the pressure wave we have developed a tendency to the formation of a beak, or, as the French observers term it, a *crochet*, at the summit of the line of ascent. This beak is due to the vibration of the arterial walls outstripping the blood-wave, and it may be increased, or more fully brought out by taking the pulse tracing with the arm elevated;† because, although in aortic regurgitation both kinds of waves are delayed, as a loose string vibrates more slowly and with larger undulations than one more tense, and an unfilled elastic tube

\* Marey, *Physiologie Médicale de Circulation du Sang*, Paris, 1863, p. 419, &c.

† Loraine, *sur le Pouls*, Paris, 1870, p. 258, &c.

is longer of reaching its maximum distention than one more nearly distended—yet by the elevation of the arm the gravitation of the blood tends to delay the arrival of the maximum of the pressure wave at its upper end, while no additional obstacle is opposed to the advance of the percussion wave. When the upward and downward lines of the beak lie in the same plane, as in the upmost of the two tracings given, our reasoning in regard to the mode of its formation is open to the objection that it may be due to the imperfection of the instrument, the initial impulse of the vibration wave having been so great as to have jerked the writing lever above the influence of the pressure wave, down to the level of which it must fall—this fall being instantaneous—before the influence of this wave can be perceived in the gradual separation of the downward from the upward line until it culminates in the summit level of the pressure wave. In like manner, we might suppose that the more distinct separation of the downward from the upward line of the beak, which is visible in the undermost of the two pulse tracings given, might possibly be due to the more rapid movement of the writing tablet in the latter than in the former case, and that, therefore, the writing lever, unduly jerked up by the vibration wave, had time in the one case to return perpendicularly on its own trace; while in the other the more rapid movement of the tablet gave its descending line a slanting direction sufficient to separate it slightly, as is the case, from its ascending trace, and thus to give it only artificially the appearance as if the vibration wave was never actually, but only relatively, separated from its concomitant pressure wave. But when we know that both of these tracings were taken by the same instrument, on the same tablet, moving at the same rate, the conclusion seems inevitable that in the one case there was a much greater obstacle to the advance of the pressure wave than in the other—that is, that the regurgitation was greater in the one case than in the other, which is precisely what really existed (*vide* Cases V. and VI.). Corroborative proof of the truth of this

statement is to be found in the fact that, whenever by increasing the pressure on the artery we destroy more or less completely the influence of the percussion wave, thus compelling the pulse tracing to be formed solely by the pressure wave ; then, whatever may be the nature of the pulse, we never have a beak formed, the pulse tracing has always a rounded summit proportionate to the degree in which the pressure has destroyed the influence of the percussion wave, and has even deformed that of the pressure wave—the latter deformity being sometimes so great as completely to efface all trace of the natural dicrotism of the pulse. Of this fact few series of pulse tracings are without notable instances,\* and the following tracings from a different disease afford a very well-marked example:—

Fig. 3.

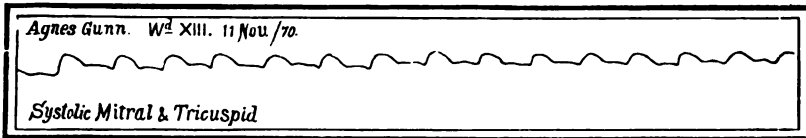


Fig. 4.



The pulse in the one tracing is much faster than in the other ; that, however, is of little consequence, the rounded summit of the one will be found to rise to a less height than the sharper peak of the uncompressed artery, and though the pulse was so feeble as to give when uncompressed but a faint tracing of the natural dicrotism (almost filiform), this will be found to be entirely effaced when excessive pressure has modified the tracing. Sphygmographic tracings of the pulse are very advantageous as revealing the local succession of events in the

\* *Vide* Marey and Loraine, *passim*.

artery ; they cannot be depended upon, however, as an index of the relation of these events to the cardiac rhythm. To determine this we must call in the aid of electricity, which has not yet been done so thoroughly as might be. Further, as by no form of sphygmograph yet invented can we insure that the pressure upon the artery shall be always the same, such tracings are of little practical value, except in so far as they indicate the regularity or the irregularity of the pulse.

The comparative longevity and comparative frequency of sudden death in aortic and in mitral incompetence have been the subject of much debate and of much variance of opinion. In regard to the comparative longevity, accurate data, involving the life history of many such patients, are unquestionably wanting, and we can only fall back with certainty upon hospital experience ; and this enables us to say that in regard to those affected with aortic incompetence, who come under treatment on account of serious disturbance of compensation, four years, reckoned from the date of admission, will include the day of death in by far the larger proportion ; while this is by no means the case in mitral incompetence. We must remember, however, that many of these cases of aortic incompetence have, from their history, evidently laboured for very many years under their valvular defect, which has only at last asserted itself as a disease, when from defective nutrition of the cardiac walls, into the causes of which I have already entered, efficient compensation could be no longer maintained. Moreover, aortic incompetence is always the result of a valvular lesion which can only be rehabilitated in the rarest instances, and then only imperfectly ; while mitral incompetence is very commonly the temporary result of perfectly curable disease,\* while ruptured compensation may be restored again and again even in its most serious and incurable forms. A due consideration of these facts enables us to say with certainty that though as

\* As in chlorosis and other forms of anæmia, &c., *vide* Lecture VI. "On Curable Mitral Regurgitation."



yet we have no sufficient data to enable us to decide as to the comparative longevity in favourable cases of aortic and mitral incompetence; yet, inasmuch as the former is always accompanied by serious valvular lesion, while the latter is not, the probability of life is very greatly in favour of a mitral lesion. Further, though we cannot estimate with certainty the value of life in any given case of aortic lesion, we may with tolerable accuracy fix the limits of vitality as lying within three months in cases of unfavourable rupture of the aortic valves, and thirty-five years in cases of disease occurring under favourable circumstances, which may vary much in character; while within these limits our prognosis must be determined entirely by the circumstances of the patient, including the necessity or not of manual exertion for procuring his livelihood, the many prospective conditions which may influence the nutrition of his body, among which his habits—temperate or otherwise, &c.,—are of the greatest importance, as well as the past history of his family, as affording a probable clue to what these habits may become; and, lastly, the family longevity of the patient, which affords a probable indication of the greater or less tendency to muscular degeneration—an indication which can never be wholly vitiated, though it may be modified by various circumstances. Further, the length of time during which the disease has probably already existed, and the present condition of the cardiac muscle, form most important elements in the prognosis. Nor must we forget, especially in the early stages of the disease, to give due prominence to the temperament of the patient, nervous excitability, whether evinced in sudden bursts of passion, or in a tendency to irregular spurts of violent exercise, being specially unfavourable—whatever conduces to irregular action of the heart being injurious in all forms of cardiac disease, but infinitely more frequently fatal in aortic incompetence than in any other. And this leads me to consider that other *quæstio vexata*, the cause of death in aortic regurgitation, and especially the frequency of sudden death in this form of cardiac lesion, compared with others. Death, whether it

arises from the loosening of the silver cord or the breaking of the golden bowl, is always a momentary and therefore sudden act; but we only so regard it when it appears sudden or unexpected relative to the apparent previous condition of the patient. When an individual, therefore, drops dead upon the street, or in a public meeting, or is found dead in his bed after retiring to rest in his usual health, he is popularly said to have died suddenly, though the sword of Damocles, in the shape of an inevitably fatal disease, has for years hung over his head ready to drop at any moment, and though his death relative to his actual state of health has been by no means so sudden as that of another hurried off by a fever or other acute disease of a few weeks' or days' duration. From this, the popular point of view, it is by no means difficult to say which of all the cardiac lesions is most likely to prove suddenly fatal. Death from any cardiac lesion occurs from syncope, and this is brought about in two ways—first, by gradually increasing asthenia, in which the aortic blood-pressure slowly fails from day to day, till at last it drops below what is compatible with life, and death ensues; second, by asystole, in which the aortic blood-pressure suddenly falls below that necessary for the maintenance of life, because the left ventricle ceases to act. No statistics are required to prove to us which of these accidents is most likely to happen in any given cardiac lesion.

When the muscular walls are enfeebled from inflammatory infiltration, fatty or fibrous degeneration, they may suddenly cease to act at any moment, either with the heart in systole or diastole—more often the latter—and with this cessation of action life comes to an end. Death from this cause may occur with or without the presence of valvular lesion; if the former, there are, as we have seen, both anatomical and physiological reasons why it should be incomparably more frequent in connexion with aortic incompetence than with any other valvular lesion. In the most extreme form of mitral constriction, enough of blood always reaches the ventricle to enable life to be maintained, if only in a feeble fashion, while no obstacle is



presented to the free action of the ventricle; in the greatest mitral regurgitation there is no want of blood in the ventricle, and provided the contraction of that organ is powerful enough, a sufficient quantity is always sent on to provide for the maintenance of life; and as the blood has in such cases always two ways of escape, ventricular systole, though it may be difficult, is always possible, except in the presence of muscular degeneration. In these two forms of disease, therefore, sudden death from asystole must be and is a somewhat rare affection, the more usual mode of death being syncope from asthenia, brought about by secondary diseases, such as embolism of the brain or lungs, or more slowly by dropsy, with or without jaundice or albuminuria; or suffocation suddenly induced by pulmonary œdema. On the other hand, in aortic incompetence the constant and continually increasing dilating pressure of the blood column acts as a permanent obstacle to ventricular contraction; and we can readily understand why comparatively trifling exertions, which tend to increase the aortic blood-pressure, and also why emotional excitement, which interferes with the nervous supply of the ventricle, and inhibits its contraction, should in very many cases be instantly followed by sudden death. The pause of a second, even with a comparatively healthy ventricle, permits the dilating power, already barely compensated, to turn the scale, and after a few feeble attempts to overcome the obstacle, the primary syncope passes into death from asystole, the heart remaining in a permanent diastole.\* And if this be the case, even with a comparatively healthy ventricle, how much more likely is this to take place when its muscular tissue is diseased, as is so frequently the case. When the mitral valve becomes incompetent from secondary dilatation, death from asystole is not so readily brought about, but it is not altogether prevented. The cavity which must then be filled to overflowing is no longer represented by the ventricle alone, but includes

\* This subject has been very fully and carefully investigated by Dr Mauriac. Vide his *Essai sur les Maladies de Cœur. De la Mort Subite dans l'Insuffisance de l'Aorte*, Paris, 1860.

also the lungs, the terminus being shifted from the mitral to the pulmonary valve. When in such circumstances the pulmonary capillaries are fully congested, any accidental interference with the regularity of ventricular action must be followed by asystole, or by artificial relief within the closed circuit, rupture of the heart or pulmonary capillaries. The mechanical cause always present in aortic insufficiency, acts with continually increasing efficiency in the direction of one or other of these two modes of death, and from the dilated and diseased condition in which the pulmonary capillaries are, and the consequent readiness with which they may be ruptured, we can easily understand that those cases of aortic incompetence which escape death from asystole, mostly die from pulmonary apoplexy, or from a combination of the two; and it is only comparatively rarely that dropsy and other secondary diseases of a serious character are established in such cases, death then occurring from asthenia. This you will see is entirely a question of animal mechanics, which statistics may confirm, but can never disprove. The mode of production and morbid appearances after death from asystole are precisely the same, whether that death has been on the street, in a public meeting, in the midst of apparent health, or after a longer or shorter period of more evident illness. In illustration of it I therefore prefer to give two cases in which the disease was known to have lasted for some time, because their history has an important bearing on the treatment of such cases, to which I shall immediately refer.

CASE V.—Isabella Stewart, aged 35, employed at paper mills, admitted on 30th May 1870, to Ward XIII., complaining of cough, which troubled her for the first time about a fortnight ago, and palpitation of the heart, from which she had suffered about eighteen months ago. Her previous health was good. Family history unimportant. Her expression was anxious, muscularity flabby, skin dusky, but naturally so; lower extremities slightly cedematous, joints normal, has never had rheumatism. Pulse 93, presenting in a well-marked



form all the phenomena of the Corrigan or water-hammer pulse. On inspection and palpation the area of cardiac impulse was found to be somewhat extended—the impulse itself, however, being somewhat feeble. There was visible pulsation in all the superficial arteries, particularly in those at the root of the neck, but by no means well-marked. The apex of the heart was found to beat under cover of the sixth rib at a distance of three inches and a half from the left edge of the sternum. At one inch from the left edge of the sternum dulness commenced at the level of the upper edge of the third rib, and extended downward to the liver dulness. Transversely, at the level of the fourth rib, dulness commenced half an inch to the right of the sternum, and extended across for a distance of five inches. On auscultation over the apex, the first sound was found to be much obscured by coarse crepitation, and also by a murmur, which, on being traced upwards, was found to have its point of maximum intensity at mid-sternum between the third and fourth ribs; the second sound is wholly replaced by a soft diastolic murmur—this double murmur is propagated into the arteries, the diastolic portion only faintly; it is also propagated with greater or less distinctness over the entire cardiac area. In the pulmonary region the pulmonic second sound is much obscured by the diastolic murmur, but on moving the stethoscope wholly off the sternum it is distinctly audible about an inch to the left of its usual position. Respirations 24; cough troublesome; sputa thin, watery, and somewhat frothy, amounting to three ounces in two hours. Pulmonary percussion normal. On auscultation, the respiration was entirely obscured by crepitation, cooing and sonorous rhonchi. Appetite gone; some thirst; urinary and other systems normal. *Diagnosis*, acute bronchitis, aggravated by the oedematous and congested conditions of her lungs, the result of aortic incompetence. *Prognosis*, grave, death being threatened by cardiac asystole. *Treatment*—full diet, six ounces of wine, dry cupping to the chest, with large jacket poultice afterwards, and the following prescription:—

R. Ammoniae carbonatis . . . . .	3i.
Tincturae hyoscyami . . . . .	3iv.
Potassii iodidi . . . . .	3i.
Tincturae digitalis . . . . .	3i.
Infusum calumbae ad . . . . .	3vi.

Sig.—One tablespoonful every four hours.

Up to 15th June, it is noted that the patient's condition had been repeatedly aggravated by attacks of pulmonary congestion and threatened asystole, from which she had been relieved by dry cupping, stimulants, and the use of the mixture prescribed. To-day there is almost no cough, expectoration nearly gone, vesicular respiration everywhere audible, pulse 88, full and strong; patient is lying quietly, and expresses herself as quite relieved. On 18th June she was up at visit, and apparently quite well; but on the 22d she had another attack of congestion, with threatened asystole, and in spite of renewed efforts, by which she was temporarily relieved, she died suddenly on 26th June. At the post-mortem examination of the body, which took place fifty-six hours after death, the heart was found more dilated than hypertrophied—it weighed 19 oz.; both ventricles were filled with dark coloured clots, of which that on the left side extended some distance up the aorta, presenting one continuous mould of the interior of the left ventricle and of the aorta. The aorta itself was slightly dilated, its valves thickened and incompetent, a considerable angular aperture being left between the edges of its cusps. Pulmonary, mitral, and tricuspid valves competent and healthy. A portion of the muscular substance of the heart was somewhat inflamed.\* The right lung was healthy both in appearance and on section, except at its lower margin, where there was a patch of pulmonary apoplexy. Pulmonic glands infiltrated with black pigment, and a good deal of pigment on the surface of the

\* This is all that is recorded in this respect in the Pathological Records. Myocarditis is always a grave complication in serious valvular lesions; unfortunately we cannot certainly detect it, and are not always in a position even to suspect its existence. I shall subsequently have occasion to relate a case of aortic incompetence in which myocarditis had an undoubted influence in promoting the fatal asystole.



lung. Left lung adherent all round, especially posteriorly, by recent lymph, in some places quite one-fourth of an inch in thickness. The lung itself is œdematous, and the bronchial mucous membrane congested. Iodine produced a slight mahogany tint of the liver, which was fatty, and presented a nutmeg appearance. Spleen healthy. Kidneys congested, but otherwise healthy; Malpighian bodies slightly prominent, but presented no reaction with iodine.

This case, then, presents a very well-marked example of death from asystole, in which compensation had apparently never been fully developed, but in which the death was certainly hastened by inflammatory complications.

In the following case the asystole was the result of long-continued disease, which had been fully compensated, but the compensation of which was accidentally ruptured. Even in his case, therefore, death was a preventible accident, though the unavoidable end was not long forestalled.

CASE VI.—William Yorkston, a blacksmith, aged 54, admitted to Ward V., 14th November 1870, complaining of orthopnoea, cough, expectoration, occasional pain in the chest, and swelling of the legs. About six weeks ago he caught cold after prolonged exposure to heat at his work, and this cold was aggravated by a fresh exposure about a fortnight ago. Has always enjoyed good health, has never had rheumatism; but for the last three or four years has been drinking heavily, and working hard. His father and mother both died of cholera. The patient was tall and muscular, his expression anxious, his face pale, his lips livid, his skin and joints normal, his legs, from the knees downwards, œdematous. Radial pulse jerks slightly, but in by no means a marked manner, beats 90 per minute. The jugular veins pulsate visibly, the right carotid slightly, and the left not at all. The cardiac impulse is diffused and feeble, the apex beating between the sixth and seventh ribs and diffusely. Percussion dulness at one inch to the left of the sternum commences at the upper border of the third rib, and extends down to the liver dulness. Trans-

versely on the line of the fourth rib, dulness commences half an inch to the right of the sternum, and extends across for a distance of five inches and a half. In the mitral area a murmur is heard to replace the first sound, but the second sound is audible. On tracing the systolic murmur upwards along a line joining the mitral and aortic areas, it is found to get gradually fainter on nearing the sternum, and again gradually to increase in loudness till it reaches its second maximum over the sternal end of the second rib on the right side; in this position the second sound is entirely replaced by a loud but soft diastolic murmur, the systolic portion of the murmur alone is propagated into the carotids. In the pulmonary region a second sound is audible, but, obscured by the diastolic murmur, it becomes more distinct on moving the stethoscope along the second interspace till its edge is quite off the sternum. At the lower end of the sternum the systolic murmur is very loud and distinct, and wholly replaces the normal sound of the tricuspid. Respirations 26 per minute; cough hard and frequent; sputum watery, frothy, and small in quantity, no blood. Percussion of lungs normal. On auscultation, sonorous and sibilant rhonchi are heard over the chest, partly obscuring the vesicular breathing. Appetite defective, bowels costive, urine scanty, no albumen. Patient sleeps none at night. He was ordered full diet and four ounces of whisky daily.

R Tinct. scillæ,  
,, digitalis, āā ʒii.  
Aque cassiæ, ʒvss. M.

Sig.—One tablespoonful every four hours.

R Elaterii gr.  $\frac{1}{4}$ , Ft. pil.—One such to be taken every four hours till the bowels are freely moved. To have twenty grains of chloral every night at bedtime.

On 21st November it is noted, patient slept last night in the recumbent posture for the first time. His dyspnoea is now comparatively trifling, the cough gone, the respiration free, the œdema of the legs entirely gone, his cardiac impulse



stronger, the pulse more markedly jerking, only 80 per minute; it is delayed the full half of a cardiac revolution beyond the apex beat; the superficial arteries pulsate now in a marked manner, the jugular veins less distinctly. Over the mitral and tricuspid areas the systolic murmur is not so loud as formerly, and much less distinctly localised. He was kept some time under observation, and then discharged much improved. On the 18th of August 1871 he was re-admitted, and died suddenly next day. At the post-mortem examination the body was well-developed; skin and conjunctivæ of a yellowish colour. The heart was greatly enlarged, dilated, and hypertrophied, weighing thirty ounces. Both ventricles contained large black clots. The aortic valves were atheromatous and incompetent, the aorta itself greatly dilated. The mitral and tricuspid valves both admitted five fingers. The right lung was very dark in colour, greatly congested, and apoplectic; it weighed two pounds two ounces. The left lung also contained an apoplectic clot, and was covered with recent lymph. The liver weighed three pounds, was fatty, and its ducts congested and obstructed by catarrhal mucus. Kidneys enlarged and congested.

These two cases illustrate very well the condition of the heart which is found after death from asystole, both ventricles, but especially the left one, being dilated and filled with a black clot extending up into the aorta, death occurring from a paralysis of the ventricular muscle, brought about by over distention. That is the mode in which death in aortic incompetence primarily invades the heart. As we have seen, it may be brought about in many ways. Our object is to prevent it, and in obviating it we shall also in a great measure be able to prevent many of the secondary causes of death, or at all events delay their fatal result.

In Case V. it is noted that the first sound over the mitral area was obscured by a systolic murmur, which had its sole focus of maximum intensity in the aortic area. This was

ascertained by tracing the murmur upwards along a line joining the mitral and aortic areas, and the results are graphically shown in the accompanying diagram (fig. 11).

In Case VI. the systolic murmur is stated to have two foci of maximum intensity; that is, that in tracing it upwards from the mitral to the aortic area, it was found first to decrease in intensity and again to increase, as shown in the accompanying diagram (fig. 12).

This is the method to be pursued in attempting to determine whether we have to do with a single systolic murmur of mitral or aortic origin, or with a double murmur originating

Fig. 11.

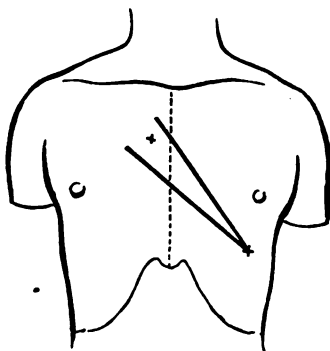
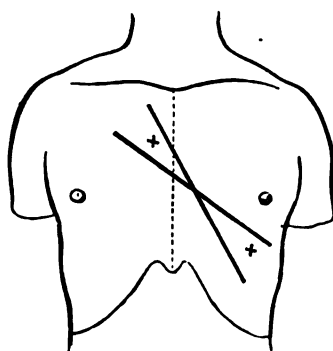


Fig. 12.



at both orifices; and these diagrams represent graphically the results we obtain in tracing the murmur from one orifice to the other in two of three hypothetical cases. Of course, when the systolic murmur is purely of mitral origin, the graphic representation would be exactly the reverse of that depicted in diagram 11.

In the treatment of aortic incompetence, we must carefully distinguish between those in whom the disease is only to be recognised by signs, and those in whom it is revealed by symptoms. In the former class the disease is dynamically compensated and is mute, in the latter the compensation is ruptured or incomplete. Those in whom the disease is only revealed by signs require no special cardiac treatment, they

or their friends must be cautioned against over exertion or over excitement, especially against any sudden and violent exertion, they must be well fed, protected as far as possible from cold or any risk of chill, and stimulants had better be altogether avoided, or only used as stimulants *pro re nata*, as their constant use invariably tends to weaken the heart. Apart from these cautions the patient must be left pretty much to his own devices, as continual watching, while it embitters his life, is no safeguard against sudden asystole, which is the only risk he runs. In other respects his health is likely to be very fair, and the only tonic advisable is one or other of the more assimilable preparations of iron, such as the wine of iron, the phosphate of iron, Parrish's or Easton's syrup, with small doses of arsenic, all of which are admirable hæmic tonics, while the arsenic seems to be a special tonic to the cardiac muscle. Any treatment likely to depress the vital powers must be avoided, and should laxatives be required, only the mildest varieties must be employed.

But whenever the compensation is ruptured from any cause, and we have symptoms superadded to signs, then we require to act more energetically; and should the disease be not too far advanced, our energetic action will often be attended by the happiest results, compensation will be restored, and life prolonged, often for many years.

In aortic incompetence the most injurious effect is produced by the dilating force of the column of arterial blood, which, according to Pascal's law, acts in proportion to its height and the area of its basis, and our primary object is to reduce as far as possible both of these elements. First of all, therefore, we put the patient to bed, and endeavour to get him as nearly recumbent as possible, so as to diminish the height, and consequently the distending power, of the arterial column. With this view we also deaden his sensibility by the administration of chloroform, chloral, or morphia, either by the mouth or subcutaneously. The orthopnoea, which is so distressing



a feature of this disease, has reference solely to the congested and œdematous condition of the lungs, and is established by nature to give the patient's respiratory muscles a better purchase in elevating the chest walls. A wise physician knows that however dangerous the condition of the respiration may be, the patient runs much more risk from the state of his heart. He does not argue the point with nature, but he calms her instinctive fears with the means at his command, and he feels that he has gained a victory for science when he has got the patient fairly recumbent. The inexorable mechanics of the circulation are then appeased, and we have leisure to attend to the physiological part of the difficulty. So intricately involved, however, are all our vital actions, that as we cannot appease the mechanical part of the difficulty without advantage to the physiological part, so also we cannot remedy the physiological portion without also benefiting the mechanical portion. It is advantageous, therefore, to carry out both parts of our treatment at once ; and though to the superficial thinker such a plan has the appearance of an empirical treatment of symptoms, a deeper knowledge of mechanics and of physiology, and of their mutual interdependence, will show that we are really employing scientific means according to scientific method, and that we apparently thwart nature only the better to secure the ends she aims at. While we therefore attempt, by getting the patient down, to lower the height of the distending column, thus at once relieving the cardiac circulation and diminishing the pulmonary congestion, we simultaneously endeavour to procure a similar relief by diminishing the area of the base of the arterial column, and this, so far as our present therapeutical armamentarium extends, we can only do by means of one drug—digitalis. It is not so long since this drug was regarded as a pure sedative to the heart—the opium of the heart, it was euphemistically termed. Now, thanks to experimental physiology, we certainly know that its action is widely different from that of opium, and greatly superior to it so far as preservation of life



in this disease is concerned.\* It is still regarded by many as not merely useless, but positively dangerous, in the disease of which I speak—aortic regurgitation;† and yet there is no other disease in which this drug is of more value, and no other in which its curative action can be more efficiently demonstrated than in this. In very large doses digitalis is employed as a sedative in many diseases, such as delirium tremens, pneumonia, &c., but experimental physiology has shown us that such a method of inducing what our American cousins call sedation is an extremely dangerous one, as it depends upon the fact that the stimulating blood-supply is cut off by an extreme degree of ventricular contraction, which falls just short of the ordinary fatal result of digitalis poisoning—death with the heart in systole. A full dose of digitalis maintains, however, its sedative action for some time without by any means proving fatal. The almost fatally contracted condition of the ventricle is not, therefore, the result merely of one or two energetic contractions which again relax, but is the result of a tonic contraction of the ventricle, which is excited only to a certain pitch by a certain dose of the drug, and beyond this it does not pass unless the dose is increased, nor does it greatly relax for some time, till the effect of the drug passes off. The ordinary employment of digitalis teaches us the same thing. If we gradually increase the dose at regular intervals, or if, employing a large dose at first, we persist in its use, similar phenomena occur. First we have a gradually increasing fulness and firmness of the pulse-beat and of the ventricular systole, and then a falling off of both—smallness of pulse, feebleness of heart-beat, irregularity, and, finally, fatal syncope with the heart in systole. There is a regular

\* For very many years I have been in the habit of employing digitalis as a cardiac stimulant, but it is only since the results obtained by experimental physiologists have been published, that I have felt justified in using it with that freedom necessary to secure its most brilliant results.—*Vide Ed. Med. Jour.*, Feb. 1870, p. 743.

† By none more definitely than by Dr Fothergill, whose work on digitalis has done more to popularise the scientific use of that drug than any other I am acquainted with.—*Vide On Digitalis*, London, 1871, p. 52.

gradation between these two extremes. So long as we employ small doses we may go on administering them daily for years—as is often done in mitral disease—without any fear of untoward result, without any dread of cumulative action. But the instant we employ large and powerful doses, the difficulty of regulating the action of the drug becomes extreme, and we require to watch its action very carefully, and suspend it on the very earliest indication of its poisonous action. The useful employment of digitalis in aortic incompetence is purely a question of dosage; in a few months or a few years we shall be able to regulate it with much greater nicety than at present, with more comfort to ourselves and more safety to our patient; at present we must risk something for the sake of an otherwise unattainable benefit. What we desire to produce in each case is just such an amount of tonic contraction of the ventricle as shall rather more than counterbalance the dilating power of the arterial column. If our patient is obliged to be upright, a larger dose will be required than if we can lay him flat; and the larger the dose required, the more carefully it must be watched. The dose must be accommodated to the circumstances of the patient, and regulated by them, as well as by his idiosyncrasy—for some are more susceptible to the action of the drug than others. In the employment of a drug where accurate dosage is of so much consequence as in this, it is of importance to have a preparation of uniform strength, and in this respect I have found the ordinary tincture all that can be desired. The ordinary infusion is not so uniform, nor is the crystallised digitaline quite so reliable. The latter is very convenient for subcutaneous injection when rapid and immediate action is necessary, but it cannot be so certainly relied upon as the tincture. In the cases already related, the doses of the tincture have varied from five to ten minims every four hours; but I have frequently doubled or tripled these doses, and sometimes even gone beyond that with benefit—in fact, though commencing, as you have seen, with small doses (five minims) of this drug, and trusting somewhat



to other tonics, I now trust mainly to digitalis, giving fifteen minims up to half a drachm of the tincture every four hours; and in one very remarkable case of persistent threatening of complete asystole I was only enabled to get the patient out of the infirmary and sent home, a distance of a hundred miles, by the continuous use of half-drachm doses of tincture of digitalis every two hours for several days.\* The use of digitalis is always accompanied by greater or less increase of the flow of urine. So long as this keeps up we are—I speak from experience—quite safe to continue its use. When employing moderate doses of digitalis, the flow of urine may halt or diminish without fear of untoward result; but in using large doses we must look upon the mere halting of the flow of urine as an indication to us to watch the pulse with great care several times a day, and if at any time it commences to thump or to falter, or if nausea be induced on movement, we ought at once to pretermitt the use of the drug. With this precaution I have never found the use of the drug in this disease, even in these large doses, productive of anything but relief, a relief unattainable by any other means I know of. Increased experience has only confirmed my faith in the use of large doses of digitalis in serious rupture of the compensation in aortic disease. I object, however, to the quantity of spirit consumed in giving large doses of the tincture, and latterly have had an infusion prepared containing fifteen grains of digitalis leaves in each ounce of water, five times the strength of the present infusion, and of this I give from half an ounce every four hours to one ounce every two hours, with perfect safety, and often with very remarkable results. I shall speak more fully of this in a subsequent chapter. After the heart has been sufficiently acted upon by the large doses of digitalis to avert the threatened asystole, it is advisable to stop it for a day or two, and then to brace up the heart by tonic doses twice a-day or oftener. For this purpose Nativelle's digitaline is very use-

\* Agnes Butler, admitted to Ward XIII. 19th October, discharged 16th November 1872.

ful, as though a little irregular in strength it disturbs the stomach less than other preparations. Where there is much starting from sleep, the *subitanea excitatio e somno* of the older authors, it dies away under the tonic influence of digitalis in a few days, and of itself does not require the use of morphia, though always relieved by it. Now and then it happens that mental aberration of a more or less violent character occurs in the course of aortic disease, there can be no fixed rule of treatment for such aberration which must always depend upon its physical basis. Some few may depend upon cortical embolism, most of them, however, seem to be induced by anæmia; and in those which have occurred to myself, full hypnotic doses of morphia hypodermically injected have been attended by the best results, and the mental quiescence thus induced has been confirmed by the use of digitalis in appropriate doses. Iron is always a risky addition to digitalis, as it is very apt to disturb the stomach and cause sickness. When urgently required I prefer the tincture of the acetate to the perchloride, but it is much better to avoid the combined use of these drugs. In such cases iron never is of so much consequence as digitalis, and a good supply of animal food may fairly enough replace it. The liquor arsenicalis stands in a different category; it is not only a hæmic tonic, but it is a special tonic to the cardiac muscle, and in moderate doses rarely disturbs digestion. In the advanced stages of aortic incompetence the compression to which the ventricular muscle and its vessels are subjected give rise, as we have seen, to mal-nutrition, especially of the muscoli papillares. Neuralgia is said to be the prayer of the nerves for better blood; hence neuralgic pain is a frequent accompaniment of this disease. In all forms of cardiac angina arsenic seems to relieve pain, and in this form it certainly acts with great benefit. At present we can only employ it empirically; but it is not so long since digitalis was only employed for a similar reason, and we confidently look forward to a time when the use of arsenic in cardiac neuralgia shall be justified by accurate physiological reasons.



Of course it is occasionally advisable to conjoin the use of these remedies with that of others possessing certain other specific actions, such as diuretics, purgatives, &c. ; but these are to be used *pro re nata*, with reference to the individual case, and not to the disease generally. All such patients require to be nutritiously yet moderately fed, because the defective aortic pressure reacts injuriously on the gastric and hepatic secretions, and limits both their supply and their efficiency. Ordinary alcoholic stimulants may be of great use in such cases, moderately supplied, but their employment must be watched and regulated. They are only of temporary value to tide over a weakly period, and by no means possess the permanent value of such special cardiac stimulants as digitalis, arsenic, or even iron.

## LECTURE IV.

ON THE MURMURS AND OTHER PHYSICAL SIGNS DISTINCTIVE OF  
MITRAL STENOSIS.

GENTLEMEN,—Were I to ask you to tell me what sign you suppose to be most distinctive of disease of the mitral valves, probably nine out of every ten would, without hesitation, reply, A systolic murmur loudest at the apex. Yet this, though the belief of a large proportion of medical men, is far from being the truth. A systolic apex murmur is by no means always a certain proof of any positive derangement of the cardiac mechanism. It is said, as you are aware, that such a murmur may be of exocardiac origin, and the valves may in this case remain healthy, and their action perfect, in spite of the persistence of a murmur having the character described. It is even said that a murmur of this kind may be of endocardiac origin, and yet the valvular mechanism remain uninjured; and though such murmurs are of very doubtful occurrence, still they are possibilities which we must never forget in estimating the probabilities in favour of our diagnosis. But systolic apex murmurs originating exocardially, or even endocardially, apart from valvular derangement, are not of course associated with regurgitation backwards into the auricle, and are free from any of the signs and symptoms dependent on that accident; yet, even though regurgitation be unequivocally proved to exist along with a systolic apex bruit, mitral disease or deformity is not therefore

a necessary consequent, because in a not inconsiderable proportion of cases in which both of these phenomena are present the mitral valve is nevertheless perfectly healthy. In fact, the mitral valve may be free from disease, and the auriculo-ventricular opening perfectly natural and undilated, and yet regurgitation may, and often does, take place. How this may be I shall take another opportunity of explaining ;\* it is sufficient for the present to state the fact that, even when mitral regurgitation is clearly established, it is no positive proof of disease of the valve. On the other hand, there is a murmur which is so invariably associated with disease of the mitral valve that, when once heard, it may be conclusively accepted as a positive proof of the existence of a permanent deformity, even though the murmur itself should subsequently disappear, as it frequently does. This murmur, which is pathognomonic of mitral stenosis, has been termed, *par excellence*, the presystolic murmur. It is well to remember that this is a misnomer. As the term is, however, a convenient one, and has been long attached to this special murmur, it is perhaps right to continue its use, provided always we recollect that it is merely conventional, and not strictly accurate.

The history of the presystolic murmur commences with M. Fauvel,† who, in 1843, not only described its characteristics, and amongst them its rhythm, but actually gave it the name by which it is still best known. British physicians are, however, mainly indebted for a clear understanding of this important murmur to the writings of Dr Gairdner, whose lucid

\* *Vide* Lecture VI. "On Curable Mitral Regurgitation."

† *Archives Générales de Médecine*, 4th série, tom. i. p. 1. Fauvel acknowledges that he borrowed the term "presystolic" from M. Gendrin, and upon this has been built the theory that we owe to M. Gendrin our knowledge of the murmur and its cause. Gendrin's claims to the origination of the theory of this murmur may be very well disposed of in his own words: "Quand le bruit est *prediastolique*, on peut penser qu'il existe un rétrécissement d'un des orifices auriculo-ventriculaires, on peut l'affirmer s'il existe en même temps un doublement du bruit de percussion diastolique."—*Leçons sur les maladies du cœur*, Paris, 1841, tom. i. p. 111.

statements and admirable diagrammatic representations were first published in 1861. In Edinburgh this murmur is of such frequent occurrence, so generally recognised, and the recognition verified every session by the discovery of the predicted pathological condition, that it is simply incomprehensible how its existence is still ignored by some even of the most esteemed members of our profession. Not, perhaps, that it is altogether ignored, though that sometimes happens—but its true character is denied, which is perhaps worse.

In estimating the nature of any murmur supposed to be of valvular origin, you are aware that it is absolutely necessary to be precise in ascertaining two facts regarding it: the first being the position on the cardiac area at which it is most distinctly heard, and the second its rhythm—that is, its proper relation to the several acts which constitute a cardiac pulsation. These points being determined, the ascertaining of the lesion upon which the murmur depends is a simple matter of ratiocination, in which it seems hardly possible to err. Now, the murmur I speak of has a fixed position in which it is most distinctly heard, viz., over what has been already described to you as the mitral area—that is, within a circle of about an inch described round the point where the apex impinges as a centre. It is not much propagated in any direction, and though the educated and experienced ear can readily detect the alteration of the first sound produced by the presence of this murmur, wherever the heart-sounds can be heard, yet it is only over a very limited area that it is audible as a true murmur, being as a rule only indistinctly audible above the third rib, while its distinct propagation is, as a rule, almost equally limited in every other direction. To give you some idea of this limitation of propagation, I may mention that there is now in the wards a lad of eighteen with a murmur of this character, so loud and rough that, on his first admission, it was easily recognised through three shirts (two of them flannel), a waistcoat, coat, and topcoat; yet this murmur, which I especially investigated to determine the



area of propagation, is not distinctly audible above the third rib, nor below the middle of the sixth interspace ; nor farther to the left in the nipple line than a line descending perpendicularly from the anterior border of the axillary space ; while on passing to the right it is already less rough at the left edge of the sternum, and is quite lost half an inch beyond its right edge.\* But though I give this as an excellent illustration of the remarkable limitation of propagation, even in an exceedingly rough and loud presystolic murmur, you are not to conclude that this murmur is always limited to so small an area, because exceptions do occur, though these are certainly much rarer in regard to its propagation than in regard to its character. In accordance, therefore, with the laws of the propagation of murmurs already laid down, this position of audition, as we may term it, stamps this murmur as of mitral origin, and even hints its probable rhythm to the intelligent mind. The rhythm of a murmur is, as you are aware, its relation to the several physiological acts which constitute a complete cardiac pulsation—that is, which occupy the time comprised between two consecutive apex beats. During this period we have the ventricular systole, synchronous with the apex beat, the first sound, and the first silence ; the ventricular diastole, synchronous with the second sound, and the second silence ; and lastly, the systole of the auricles, which coincides with the latter portion of the ventricular diastole, and immediately precedes the ventricular systole running into it.† In timing any murmur, therefore, we must take the greatest pains to discover whether it takes the place of either the first or the second sound, or if not, then we must ascertain what is the accurate relation of the murmur to these sounds, which of them it precedes, or which of them it follows, and at what interval. Now, in this

\* W. C., Ward V.—The murmur in this case passed further to the right than usual. He has since died. His case will be found detailed further on, where the reason for this will appear. *Vide* Case IX. p. 112.

† *Vide* diagram at p. 37.

matter there is this fallacy, that both first and second sounds are not always audible together at apex or base, as the case may be, either in health or disease; and when this is the case in disease, the murmur present is apt to be taken as replacing the absent sound. This is specially the case with the murmur of which I am at present speaking, for in the mitral region this murmur is not infrequently followed by a single loud accentuated sound; and, accordingly, most students at once assume the case to be one of mitral regurgitation (systolic murmur) with accentuated second sound. The only means of correcting this erroneous diagnosis is to time both the murmur and the sound by placing our finger on the carotid artery while listening to the murmur, when the murmur will be found immediately to precede and to run up to the carotid pulse, with which the sound is distinctly synchronous. But we know that the carotid pulse is synchronous with the first sound of the heart, and precedes the second one; the sound that we hear is therefore the first sound of the heart, and as the murmur immediately precedes it and runs up to it, it occupies the time of the auricular systole; and inasmuch as the ventricular systole is the first portion of the heart's action, giving rise to audible or tangible phenomena, and is usually called the heart's systole *par excellence*, so this murmur has been termed presystolic; but, as I have already said, this is a misnomer—it is truly systolic in rhythm and character—but the systole on which it depends is that of the auricles, and not that of the ventricles; it is an auricular systolic murmur. In timing this murmur it is obvious that we must employ the carotid and not the radial pulse; for while the former is always synchronous with the ventricular systole and apex beat, the latter is even in health always delayed to an appreciable extent—one sixth of a second; while in disease, especially such as interferes with the arterial contractility, this delay is notably increased, and sometimes amounts to an entire cardiac pulsation. A reference to the radial pulse is thus always embarrassing, and

may greatly mislead; but a reference to the carotid pulse is a perfectly safe guide, provided we ourselves have senses educated sufficiently to appreciate the teachings obtainable by comparing an audible with a tangible phenomenon. As, in a pretty considerable experience of clinical teaching, I have rarely seen any mistake made in doing this, the difficulties in its way must be but slight; such as they are, they are readily surmounted by the repeated examination in this manner of hearts which are either naturally slow, or have had their action artificially retarded.

Much that is written as to the rarity or even non-existence of this peculiar murmur must surely be chargeable to inattention to this simple, efficient, and necessary diagnostic procedure. There are other murmurs besides this dependent upon mitral constriction, of which we shall presently speak; even this murmur is not always audible when its cause is present, still it is by no means infrequent, and a due attention to the means described for ascertaining its rhythm and position of audition ought to leave no doubt as to its real character, and just as little as to its frequent occurrence. The peculiar position—in the cardiac rhythm—of the presystolic murmur has given rise to an arbitrary regarding it as systolic or diastolic, according to the views of the observer. A simple reference to the carotid pulse, while we listen to the cardiac sounds, is sufficient to correct this, and to prove as convincingly as anything sublunary can be proved, that this murmur is preceded by the long pause, and cannot therefore be diastolic, while it precedes the carotid pulse, and therefore both the apex beat and the short pause, and cannot therefore be systolic, but that it occurs just before the external signs of the ventricular systole. This murmur, therefore, commencing just at the end of the ventricular diastole, and preceding its systole, must occupy the time which every physiologist knows to be that of the contraction of the auricle, and must of necessity be auricular-systolic. The term post-diastolic, applied to this murmur by some authors, apparently with the



view of indicating its position in the cardiac rhythm, and yet avoiding a direct avowal of its auricular-systolic character, is misleading and unnecessary. The ordinary conventional term, presystolic, conveys the same idea in quite as vague a manner, and has the advantage of long prescription in its favour, besides being more distinctive, in the sense of conveying the idea that there is something peculiar in its character, not to be found in any strictly diastolic murmur. Other observers, among whom I may mention Dr Andrew Barclay of London,\* Dr Leaming of New York,† and Dr Frank Donaldson of Maryland University, U.S.A.,‡ have argued, with more or less plausibility, that this presystolic murmur is not produced by the systole of the auricles at all, but by the early and soundless portion of the systole of the ventricles. To all such arguments it might at first sight seem practically sufficient to reply, that so long as what is called a presystolic murmur is duly recognised as an infallible sign of mitral stenosis, it is a matter of no importance whether we regard it as auricular-systolic or ventricular-systolic in rhythm. But, unfortunately, though an adept may easily enough recognise a presystolic murmur without timing it, it is quite impossible to teach others what we mean by the term presystolic murmur, without directing very special attention to its rhythm. Any want of clear and accurate ideas in this respect leads inevitably to the supposition that the discrimination of a presystolic murmur, which is really one of the simplest problems of cardiac diagnosis, is one which is not only most difficult, but often almost impossible of solution; nay, want of precision in this matter has led even a man of so much experience as Professor Flint to describe as presystolic, murmurs which must have been diastolic in rhythm.§ To

\* The *Lancet*, March 1872, pp. 283, 353, and 394.

† *New York Journal of Medicine*, June 1868.

‡ Paper from Author. Read before the Medical and Chirurgical Faculty of Maryland, April 1874.

§ *A Practical Treatise on the Diagnosis, Pathology, and Treatment of Diseases of the Heart*, Philadelphia, 1870, p. 207.



avoid falling into such errors yourselves, you must be careful always to time the murmur as I have directed ; and though it would be out of place in practical lectures such as the present to enter into any long physiologico-pathological disquisition, it may be well to remind you that the auricular-systolic character of the presystolic murmur is proved not only by its position in the cardiac rhythm, but also by its exact coincidence with the pulsation of the auricle so often to be felt above the fourth rib in such cases.\* This murmur is further a direct murmur, accompanying the onward current of the blood in its natural course through the heart, forcibly produced by muscular contraction. We shall presently see that there are other murmurs, occurring at the mitral valve, direct as to the natural current of the blood, but having all the softness characteristic of an indirect or regurgitant murmur, because they are actually diastolic in rhythm and unaccompanied by forcible muscular contraction. This, however, is not the character of the true auricular-systolic murmur ; it is short, because it sharply coincides with the contraction of the auricles ; it is also rough, because it is a direct murmur produced by forcible muscular contraction. It has been asserted by some that this loudness and roughness is entirely dependent on the amount of constriction or roughness of the upper surface of the valve. That neither this nor the directness of the murmur are the sole causes of the roughness is rendered doubtful by the remarkable manner in which this murmur occasionally disappears, temporarily or permanently, the condition of the valve remaining the same. Something in this may be due to the constitution of the blood, and

\* At p. 714 of the *Lancet* for May 25, 1872, I have entered pretty fully into the various physiological and other reasons opposed to the assumption of the systolic rhythm of the presystolic murmur. To that paper I must refer those disposed to pursue the subject beyond the practical evidence developed in the text. I may also add, that the evidence of the cardiograph is distinctly in favour of the auricular-systolic rhythm of this murmur.—*Vide* Dr Mahomed in *Medical Times and Gazette*, April 1872 ; and Dr Galabin, in *Guy's Hospital Reports*, 1875.

something also to the varying power of but that these are not all-sufficient is shown by rapid alterations in the degree of audibility of murmurs which, as well as the occasional production and conduction of other sounds, are as yet wholly inexplicable.

Various combinations of vowels and consonants are employed to represent, phonetically, the different murmurs. The murmur I now speak of, which is most susceptible of being thus represented, is *Rrrrb* or *Vōōt*, when vocalised, conveying almost identical with those produced within the chest, the very accent with which these symbols may be made to represent the changes within the different murmurs, or in the same murmur, the last consonant in each symbol being of the apex beat.

Furthermore, sounds are only audible in many cases these are to be felt as well as heard. The passage of a carriage along a street produces a perceptible vibration in the houses in it; and if we strike a tumbler of water, it produces a musical note, the vibrations of which are readily felt on placing a finger on the edge of the glass. The sound ceases at once when we stop these vibrations. Moreover, the sounds produced by friction of the fingers along the edge of glasses partially filled with water are only to be felt as vibrations, but may be seen in the motion of the water within them. In like manner, the vibrations which give rise to cardiac murmurs may frequently be felt the finger at their point of origin; and as they are felt the louder and rougher the murmur is, and the fewer murmurs, if any, so rough as the presystolic murmur frequently than any other gives rise to a distinct vibration. This feeling of thrill is termed *cataire*, or purring tremor, it is rarely absent in mitral stenosis, being sometimes to be felt in the m

of any remarkable hypertrophy of the heart shown in the following case:—

Margaret Ross was an inmate of Ward XIII. Suffering under general dropsy, depending on kidney disease (the urine was of a pale, watery, white), for which she was repeatedly bled. The chief concern now is, that she had a marked rough presystolic murmur, accompanied by a loud systolic murmur at the apex. On dissection her kidneys were found as expected, her heart was somewhat enlarged, weighing ten ounces, with milk spots on its anterior surface. The left ventricle was in a state of concentric hypertrophy; its walls were considerably thickened (chiefly on the kidney disease); the segments of the mitral valve were united and thickened throughout, but not contracted; their surfaces were perfectly smooth, and the opening, which was so contracted as to be the size of the middle finger, there were one or two small openings; the cordæ tendineæ were contracted and the aortic valves were competent and the heart apparently natural.

A marked case, in which the post-mortem examination confirmed the diagnosis during life, as, I may say, in similar cases.

She had a presystolic murmur so loud that I detected it as a measure of the extent to which it would be propagated. In mapping out the murmur, I found it to extend so much that the thought struck me, Is this case not only a mitral but aortic? I dismissed the idea as in the case of the great propagation of the presystolic murmur. The question is, however, a very important one.

given rise to many unlucky *contretemps* not very creditable to practitioners of medicine. For instance—

CASE VII.—I well remember a case in which a patient with a disappearing presystolic murmur possessed quite a bundle of certificates from medical men, one-half of which testified that he laboured under organic disease of the heart, while the other half certified, equally strongly, that he was altogether free from cardiac disease. He ultimately died, and the mitral valve was found to be deformed, the opening constricted, and the appendix of the left auricle filled with an organised clot. Of course in every such case there are various subsidiary phenomena which tend to prove the persistence of the cardiac lesion, apart from the existence of a murmur; but of these I shall presently have to speak more at large. Let this case be a warning to you to pay more attention to those subsidiary phenomena, when you undertake to confirm or controvert the deliberate opinion of a competent medical man, and to avoid basing your opinion as to the existence of cardiac disease on the mere presence or absence of a murmur, as either phenomenon may occasionally mislead, though, as I have already said, such a murmur as that just described never does.

The condition of the valve usually associated with the murmur described, is that which has been termed the diaphragmatic valve, in which the two segments of the mitral are united and stretched like a diaphragm across the auriculo-ventricular opening. The valves themselves are thickened, especially at their edges; sometimes they are almost cartilaginous in character, and their surface is usually smooth, though occasionally their edges have a few small and frequently calcareous vegetations attached. The central opening may vary from a small buttonhole, into which the point of the little finger can scarcely be inserted, up to an aperture not very much less than normal. Even where the murmur has been very loud, rough, and persistent, the constriction is not necessarily great, nor the valve rougher or denser than usual; roughness of the murmur is quite independent of roughness of



the valve, or even of any remarkable hypertrophy of the auricle, as is very well shown in the following case:—

CASE VIII.—Margaret Ross was an inmate of Ward XIII. for a whole year, labouring under general dropsy, depending on kidney disease (large white), for which she was repeatedly tapped, &c. What chiefly concerns us now is, that she had a persistent and well-marked rough presystolic murmur, accompanied by a thrill at the apex. On dissection her kidneys were found diseased as expected, her heart was somewhat enlarged, weighing fifteen ounces, with milk spots on its anterior surface; the left ventricle was in a state of concentric hypertrophy, but besides this its walls were considerably thickened (this depending probably on the kidney disease); the segments of the mitral valve were united and thickened throughout, but chiefly at their margins; their surfaces were perfectly smooth, and on the edges of the opening, which was so contracted as only to admit the point of the middle finger, there were one or two minute vegetations; the cordæ tendineæ were contracted and matted together; the aortic valves were competent and natural; the right side of the heart apparently natural.

This was a very well marked case, in which the post-mortem phenomena fully confirmed the diagnosis during life, as, I may add, invariably occurs in similar cases.

CASE IX.—William Craig had a presystolic murmur so loud and rough that I have selected it as a measure of the extent to which such murmurs could be propagated. In mapping out the propagation of his murmur, I found it to extend so much further to the right than usual that the thought struck me, Is it possible that we can have in this case not only a mitral but also a tricuspid stenosis? But I dismissed the idea as in the highest degree improbable, and referred the great propagation to the loudness and roughness of the mitral murmur. The result shows that in this I was mistaken, though unquestionably there were no other symptoms present but the excessive propagation of the murmur which could countenance the former idea. The patient was anæmic, and there were con-

sequently no jugular throbbings present, and the increased transverse dulness of the heart could not of course be regarded as any definite sign of tricuspid stenosis. The patient died of latent pneumonia following, and apparently induced by scattered patches of pulmonary apoplexy (embolic pneumonia), a condition which had been recognised during life. On opening the thorax, the enormous size of the right auricle at once attracted attention. It measured inside eight inches and a half in circumference by two and three-quarters vertically, and contained a large clot, half an ounce of which was decolorised; its walls were somewhat thickened, but irregularly so, varying in thickness from one line to a quarter of an inch. The muscoli pectinati were singularly well developed. On looking down on the tricuspid valve from the auricular aspect, its segments were found matted together, and the opening so constricted as only to admit the point of the middle finger; the endocardium in its neighbourhood was slightly thickened; a few small vegetations were attached to the free margin of the valve. The right ventricle was slightly dilated; its walls not hypertrophied; the cordæ tendineæ contracted; and no distinctive trace of the three segments of the valve were to be found. The left auricle was dilated so as to admit a ball two inches in diameter; its walls were not hypertrophied; its endocardium was thickened. The mitral valve from the auricular aspect scarcely admitted the tip of the little finger; its upper surface was covered by many calcareous spiculæ; its free margins beset by a few small vegetations. The left ventricle was apparently normal both as to its walls and cavity. The aortic and pulmonary valves were competent and healthy. The whole heart weighed nine ounces and a half. The rest of the dissection is omitted as unimportant for my present purpose. The boy's illness, according to his own statement, dated only a few months back. There was no history of rheumatism in either of these individuals.

Cases illustrative of this peculiar murmur and its accompanying lesion are of such common occurrence, both in the



wards and in the pathological theatre, that no session passes in which you may not have repeated occasion to verify the statements made, it is unnecessary therefore to multiply them here.

Of course, with a mitral valve deformed in this manner, closure of its segments is necessarily impossible, and regurgitation to a greater or less extent must be present in every instance, yet a systolic bruit is frequently wanting, as in the two cases just related, while perhaps quite as frequently, as I shall presently point out, the systolic murmur is the only one distinctly audible, and in yet a third series of cases both murmurs are audible separated by the apex beat.

The pulse in this form of cardiac disease is weakened in proportion to the amount of stenosis present, and is more or less irregular,\* though not always markedly so, provided no pyrexia coexist; and the accompanying diagram may be

\* Marcy in his *Physiologie médicale de la circulation du sang*, Paris, 1863, p. 526, has expressed his opinion that a mitral constriction, when sufficiently marked, to give rise to a diastolic murmur, suppresses the irregularity of the pulse. A glance at the pulse tracings by which he seeks to confirm this view, at once explains upon what it is founded. When the stenosis is not extreme, and the compensation is good, the murmur is usually loud and rough, and the pulse tolerably full and regular. Marcy has evidently taken his illustrations and based his opinions on cases of this kind. But we see such cases only incidentally. They only come to us as patients when compensation fails; and one of the earliest consequences of this is defective nutrition of the cardiac muscle, and as its result irregularity of the heart's action and of the pulse, more marked in this form of cardiac disease than in any other, only occasionally emulated in atherosclerosis of the coronary arteries, or cases of extreme ventricular dilation, and often amounting to a veritable delirium cordis. Physiology is at one with clinical experience in this matter. Irregularity of the pulse has been noted as specially pertaining to mitral stenosis from the earliest times, as may be seen by reference to the following authorities who have attended to this matter, which a great many have not:—Adams, *Dublin Hospital Reports*, vol. iv. p. 494; Elliotson, *Lumleyan Lectures on Various Diseases of the Heart*, London, 1830, p. 22; Bouillaud, *Traité Clinique des Maladies des Cœur*, Paris, 1836, tome ii. p. 217; Hope, *Treatise on the Diseases of the Heart*, 3d edition, London, 1839, p. 376; Stokes, *Diseases of the Heart and Aorta*, Dublin, 1854, p. 192; Freidreich, *Krankheiten des Herzens*, Erlangen, 1867, 2<sup>te</sup> Aufl. s. 249; Von Dusch, *Lehrbuch der Herzkrankheiten*, Leipzig, 1868, s. 213; Gerhardt, *Lehrbuch der Auscultation und Percussion*, 2<sup>te</sup> Aufl., Tübingen, 1871, s. 284; Balthazar Foster, *Clinical Medicine*, London, 1874, pp. 103 and 320, &c; Rosenstein, *Ziemssens Cyclopædia*, vol. vi., London, 1876, p. 130.

murmur were heard gradually to increase in loudness to the upper edge of the sternum, where it is joined by the first rib, but nowhere on the right did they attain the same loudness and distinctness as on the left. A slight humming murmur was occasionally audible in the veins. For the last month the patient's urine became more scanty, uræmic vomiting and sickness were frequent, and she gradually sank, and died on the 21st December, the immediate cause of death being a pleuro-pneumonia absolutely latent, and entirely without any subjective symptoms whatever. Her cardiac symptoms remained unchanged. At the autopsy, on 23d December, the lower part of both the right and left lung were found to be hepatised, and were covered externally by a thin layer of perfectly recent lymph, presenting a honeycomb appearance. The left lung was slightly retracted, uncovering the heart to a greater degree than usual. The heart itself was purse-shaped and somewhat enlarged, its substance healthy; the aortic valves were competent, but its cusps were thickened and covered over their whole under surface by numerous vegetations; the mitral valve was thickened and contracted, scarcely admitting two fingers, and with some threads of recent lymph attached to its edge; the upper surface of the aortic segment of this valve was thickly studded with rough stumpy vegetations of varying size. The liver was healthy. The spleen weighed fifteen ounces, and on its posterior border had seven hæmorrhagic infarctions of a triangular shape, and varying from the size of a pea to that of a small bean (the results of embolism), otherwise it was healthy. The kidneys were slightly enlarged; the right weighed five, and the left six ounces. The cortical substance was lessened; the capsule natural, and when peeled off exposed a smooth organ. The intestines were congested, and the rectum and lower part of sigmoid flexure of the colon were thickened.

The next case is perhaps equally instructive, but wants the crucial decision of a post-mortem examination. I shall confine myself solely to the cardiac phenomena:—



CASE XI.—J. H., aged 18, was sent up to Ward XIII., convalescent from typhus from the fever house, on 6th June 1871. She states that two years ago she had a severe attack of rheumatism which affected all her joints, but not her chest, and lasted for four months. After her recovery she began to suffer from pains in her cardiac region, palpitation of her heart, and shortness of breath on going up stairs. Three months before her attack of typhus fever, from which she has just recovered, she first observed some degree of swelling of her feet. Since her fever the pains in her chest have been more severe, but she has no dyspnoea while lying still in bed. Her pulse is 84, rather small, steady and regular; there is a perceptible thrill over the apex of the heart. The longitudinal dulness of the heart is normal; its transverse dulness in the line of the fourth rib extends from the right edge of the sternum towards the left for a distance of three and a half inches. The apex beat is between the fifth and sixth ribs, three inches and a half from the left edge of the sternum. On auscultation over the apex beat a somewhat prolonged murmur is audible, the first portion of which is rough, and precedes the apex beat, while the latter and softer portion follows it. Between the second and third ribs on the right side there is a systolic murmur prolonged upwards, and followed by a normal though somewhat feeble second sound. Between the second and third ribs on the left side, close to the sternum, there is a systolic murmur, followed by a markedly accentuated second sound, which is immediately succeeded by a soft, blowing, diastolic murmur. This diastolic murmur ceases to be audible at a distance of two inches and one-third from the left edge of the sternum along the second interspace. Descending from the third rib at one inch from the sternum, this diastolic murmur continues audible till about the middle of the fourth rib, after which it ceases to be heard, the presystolic and systolic murmurs being alone audible beneath this point. Over the sternum this diastolic murmur is only to be heard between the second

interspace and the fourth rib. The systolic murmur is slightly propagated into the carotid arteries, as is also the aortic second sound which remains pure. During the progress of the case the ventricular-systolic murmur over the apex was frequently found absent, and the diastolic murmur at the base became more limited in its area. The patient was discharged improved on 12th August.\*

The murmurs in these two interesting cases differ considerably in some respects, and yet both belong to the same category—examples of which, though not very common, are yet by no means rare. And I shall first remark that the systolic basic murmur on the right or aortic side of the sternum, in the second case, very probably depended on a similar roughened condition of the under side of the aortic semilunar valves, which was observed in Macmurray; that the murmur was not distinctly heard in her case (the first sound in that position was only muffled) is no proof to the contrary, as these anomalies constantly happen, but, on the other hand, it is just possible that it may have been propagated from the left side, in which case it belongs to quite a different category; at the same time, had this been its only source, it would scarcely have been propagated so distinctly into both carotids, at least we should not expect it to have been so. The first point having a direct bearing on the subject in hand—the diagnosis of mitral stenosis—is the occurrence in both of these cases of a diastolic murmur, loudest at the right edge of the sternum at the base of the heart. In H.'s case there never was any doubt as to the diastolic character of the soft blowing murmur; in Macmurray's case, her ordinary pulse of 104 was reduced by digitalis and the recumbent posture to 80 before I could positively satisfy myself that her musical murmur was truly diastolic.

\* J. H. gradually developed aortic incompetence, with all its usual results. She married in 1879, had her first child early in 1880, and died under my care from asystole due to advanced aortic disease in March 1880. I was unable to procure an examination of her body.



The region of the pulmonary artery in the neighbourhood of which these murmurs are frequently heard,\* has been not inaptly termed the region of romance, because of the murmurs audible there, which have given rise to much speculation, and which nevertheless are all easily explicable. Laennec, it is well known, taught that the second sound was due to the contraction of the auricles; and Hope, though well aware of the production of the second sound by the closure of the semilunar valves, yet taught that "when the mitral valve is contracted, a murmur accompanies, and sometimes entirely supersedes, the second sound," the influence of Laennec's teaching helping no doubt to mar the excellence of his own observation. For the diastolic murmur of mitral stenosis never obscures the second sound, though it often, as in the cases just described, immediately follows it; sometimes, as in them, separated by an appreciable interval from the true presystolic murmur (fig. 3), at others including it and running right through the periods of diastole and rest up to the apex beat (fig. 2). In both classes the first portion of the murmur is distinctly soft and diastolic in character, while the latter portion has a rough and systolic character, the direction of the current of blood being the same during both, clearly showing the influence of muscular propulsion in roughening the murmur. In Macmurray's case there is positive proof that the condition of the pulmonary valves had nothing to do with the production of the diastolic murmur; the absence of any symptoms of disease of the right heart, in the other case, coupled with the great rarity of disease of the pulmonary artery, and the comparative frequency of similar diastolic murmurs in other cases of mitral stenosis, make it

\* The position of maximum intensity of this mitral diastolic murmur varies in each case; it is however usually about the sternal end of the fourth rib on the left side, though not infrequently it is heard in the mitral area, and frequently, as stated in the text, in the pulmonary area. The soft slightly *musical* diastolic murmur audible in such cases over the mitral area, or more faintly in the pulmonary area, is undoubtedly mitral in its character, and may disappear as the disease progresses, a soft *blowing* diastolic murmur when heard at or below the level of the aortic valve, is just as certainly due to commencing regurgitation through this valve, and gradually increases as the disease progresses.

most probable that this diastolic murmur in all of them depends upon the same cause. The appreciable interval which existed between the diastolic and presystolic murmurs, in both cases, makes it impossible that prolonged auricular contraction, encroaching on the periods of rest and ventricular diastole, could have been the cause of these diastolic murmurs. In the face of a continuously contracting auricle, no theory of the production of these murmurs could account for a soundless interval interposed between a soft and a rough murmur, though it might explain the gradual roughening of the murmur towards its close. On the contrary, if we suppose two different causes acting in the same direction to be at work in the production of these murmurs, we have an efficient explanation of the difference in their character. And if we further suppose the first of these causes to diminish to zero before the commencement of the second, in the normal condition of the heart, we have also an efficient explanation of the pause which is occasionally interposed between the soft and the rough murmur, and we can also perceive a very plausible reason for the occasional absence of this pause. For it must be evident, that the more nearly the mechanism of the heart approaches the normal, so much the more closely will the physiological phenomena approximate those in health. The presence or absence of an appreciable pause in abnormal conditions will therefore be a measure of the amount of abnormality existing—a measure of the obstacle to the onward flow of the blood, of the degree of contraction present; just as the presence or absence of a diastolic murmur in such cases may be accepted as a probable indication of a very considerable pulmonic blood pressure, as a most important element in its production, as no murmur can arise unless the blood passes through the constricted portion “avec un force suffisante.” \*

If we take an india-rubber bag-syringe, and, after emptying it of air, insert its nozzle into water, it will be found to fill with a rapidity proportioned to the size of the opening in its

\* *Vide antea*, p. 43.



nozzle, into which, if any particle of dirt get, the bag either fills more slowly, or, if the opening be quite stopped, it ceases to fill at all. So it is with the heart; in the normal condition of the auriculo-ventricular valves the diastole is completed at once—the blood not so much flowing freely into the ventricles, as closely following in a liquid mass the recession of their walls, for of course there is in the heart no vacuous cavity or air-filled space into which the blood can be, strictly speaking, said to flow. During the period of soundless diastole the blood continues to pass into the heart, till the rhythmic wave of systole seizes upon the auricles, which then contract upon their contents, forcing them into the already full ventricles. When from any cause the auriculo-ventricular opening is narrowed, it is certain that the ventricles must fill more slowly, and this must increase according to the amount of mitral stenosis present, until at last the stimulus of contraction is felt by the auricle before the ventricular diastole is perfectly concluded. Under these circumstances, of course, the pulmonary congestion must be very considerable, and the necessary result of that is a rise of blood pressure in the pulmonary artery, and premature closure of the pulmonary valves, with a consequent want of synchronism in the closure of the aortic and pulmonary semilunar valves, and reduplication of the second sound\*—a phenomenon of such frequent occurrence in the history of mitral stenosis as to be of considerable importance in the diagnosis of obscure cases, being in many of these a tolerably permanent and by no means a casual or infrequent occurrence. The flow of blood through the contracted valve in such cases of prolonged diastole may be altogether soundless, or it may be accompanied by a murmur which is probably always faintly musical in character, and presents none of the roughness of the auricular-systolic murmur, in which it not infrequently terminates, and this notwithstanding that both are equally direct murmurs so far as the current of the blood is concerned. It seems probable that the markedly musical

\* *Vide* Lecture I., p. 33.

character of the murmur in Macmurray's case was produced by the vibrations of the rough, stumpy vegetations covering the upper surface of the aortic segment of the mitral valve; it seems possible, however, that in many cases a distinctly humming, though perhaps less distinctly musical, character may be given to the murmur merely by the peculiar state of tension of the valve itself.

You will observe that, as in the normal condition of the heart, there is a soundless interval between the ventricular diastole and the auricular systole, so the more closely the diseased heart approaches to the normal one, the more likely we are to have a similar soundless interval between the diastolic and auricular systolic murmur. The absence of any such interval is, according to the view now taken, a proof of the great amount of stenosis present; and you will observe that Macmurray's heart bears out this view, inasmuch as the pause was distinctly marked in her, while the opening in the mitral valve actually admitted two fingers, which indicates a by no means great amount of contraction. You will also observe, that this explanation of the production of a prolonged murmur, occupying the whole of the ventricular diastole, is not only consistent with what we know of the physiological action of the heart, but also with the character of the sounds heard. It also does away with all the difficulties in the way of explaining how or when an auricle is to be filled, if we imagine that a murmur, occupying as it frequently does the whole of the diastole, is produced by continuous auricular contraction,\* the pulmonary congestion very efficiently accounting for the reduplicated second sound in the manner explained.

Murmurs such as these may be graphically represented by the second and third illustrations in diagram 1, the first exhibit-

\* Macmurray's case has already been commented on in relation to the diagnosis of substernal aneurism, at p. 709 of the *Ed. Med. Journal* for Feb. 1871. The occasional absence of the presystolic portion of the murmur in her case gives it a very striking resemblance to that of Harriet H. (case 67), in Dr Hilton Fagge's interesting paper on the "Murmurs attendant on Mitral Contraction," at p. 326 of *Guy's Hospital Reports*, vol. xvi. 1871.



ing them when they are continuous, the second when, as in the two related, they are not, part of the shading to the right of the centre being omitted to represent the pause. Phonetically they may be vocalised by *rrrrrb*, or, when softer and more musical, by *roo-oo-oo-oo-b*, and when we have reduplication of the second by *roo-oo-oo-oo-b-ta-ta*.

The systolic murmur audible in the pulmonary region is not specially distinctive of mitral stenosis. I shall describe its mechanism on another occasion.\* It is very well known that a constricted mitral valve is not always diaphragmatic in character, but is occasionally funnel-shaped, the opening being at the apex of the cone which dips into the ventricle. I have no dissections of such cases to relate, but have occasionally had cases which presented characteristics which I conceived to be due to this form of valve—these are prolongations of the murmur, followed by a loud and accentuated first sound. It is such cases which are so apt to be mistaken at first hearing for a systolic murmur with an accentuated second sound; a reference to the carotid pulse at once corrects this misconception. The murmur in this case is aptly vocalised by the sounds *roo-oo-oo-lúp*, and the perfect closure of the valve has seemed to me readily explicable by the ease with which the sides of a long and somewhat loose valve, with a small opening, may be collapsed one upon the other.†

Such, then, are the murmurs specially distinctive of mitral stenosis; I shall subsequently take up the physical signs diagnostic of this affection when no murmur, or when only a systolic one, is present. The mere addition of an apex systolic murmur to those described as distinctive of mitral stenosis in no way affects the diagnosis, and its consideration is therefore omitted.

\* *Vide* Lecture VI.

† One of the best marked of these cases, with a very persistent prolonged murmur and sharply accentuated first sound, whom I had under observation for many years, at last developed an ordinary short, rough, presystolic murmur, which since then has frequently varied in character. He still lives, but the change in his murmur clearly throws a doubt on this means of detecting a funnel-shaped valve, for which there is, I believe, no reliable pathognomonic sign.

## LECTURE V.

ON THE MURMURS AND OTHER PHYSICAL SIGNS DISTINCTIVE OF MITRAL STENOSIS—CONTINUED, INCLUDING THOSE OF INCURABLE MITRAL REGURGITATION.

GENTLEMEN,—You have learned, I hope, from the previous lecture, that the essence of the cardiac affection in mitral stenosis is obstruction to the onward current of blood at the mitral valve. We deduce this from the fact that the only murmurs distinctive of this form of valvular lesion are those which, from the position at which they are best heard, are known to originate at the mitral valve, and which, from the time during which they are heard, are recognised as occurring during the ventricular diastole, but especially during that part of it occupied by the auricular systole. Now, these murmurs, as I have already described them, are quite distinctive of mitral stenosis, and when once the existence of one or other of them has been recognised, disease and deformity of that valve may be predicted, with a perfect certainty that it will be found after death. Unfortunately these murmurs are not always present, and sometimes, though occasionally audible, they are not always to be heard at the period of examination, that very time when it is of the greatest importance that a cardiac lesion should be recognisable. In most cases the diastolic portion of the murmur is entirely wanting, and in many even the auricular-systolic portion seems, if we can trust a hospital history of two or three years at least, never to have been



audibly present at all; but, besides this, each portion of the murmur may vanish temporarily for a longer or shorter period, and this sometimes in a most remarkable and unaccountable manner.\* Further, as a necessary result of the condition of the valve in the most usual form of mitral stenosis (the diaphragmatic valve), we have regurgitation of the blood backwards into the auricle, occasionally revealed by a systolic murmur in the auricular area.† In perhaps the larger proportion of those cases where the presystolic murmur is present, this regurgitation is unattended by any murmur in the mitral area; in a smaller proportion we have both the auricular-systolic and the ventricular-systolic murmurs constantly present in this area; in a much smaller proportion we have these murmurs presenting themselves in an irregular fashion, both murmurs being present for one or two beats, the auricular-systolic or the ventricular-systolic, chiefly the latter, recurring alone for an irregular number of intervening beats; while in a considerable number of cases, even when very great stenosis exists, we have the ventricular-systolic murmur alone and constantly persisting. Moreover, when the patient is enfeebled, temporarily or permanently, a ventricular-systolic murmur is often found to replace the pre-existing auricular-systolic murmur, which may again recur should the patient's strength improve.

Such, then, are the variously complicated forms of murmur which may be heard in cases of mitral stenosis. It would be easy to narrate to you cases illustrative of each variety, but it is quite unnecessary, and would only oppress your memories with unnecessary details. So far as murmurs are concerned, those only are distinctive of mitral stenosis which have been

\* In *The Practitioner* for Dec. 1873, p. 401, Dr Gowers has directed attention to the influence of posture in altering the character of these murmurs, and has pointed out that in some the presystolic murmur is absent in the erect posture, and only audible in the recumbent posture. This only holds good in a minority of such cases; in the greater number the presystolic murmur is most distinct and often only to be heard in the erect posture, and after exercise.

† *Vide* Lecture VI.

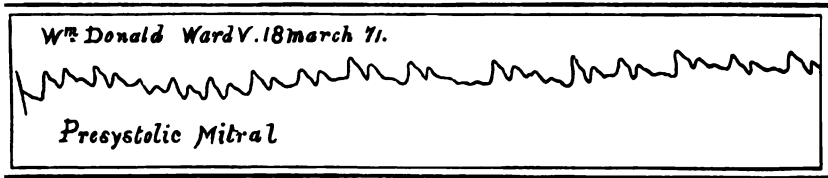
already described, the essential part of them being the presystolic murmur; whenever that is recognised as persistent, or can even be picked out as occasionally recurring amid a complication of other murmurs, the case is clear and wants no further proof. When, however, no presystolic murmur, or no murmur at all, can be detected, we are forced to unravel the case by a careful investigation of the various subsidiary phenomena evolved during the cardiac action, as well as of the relative condition of the different cardiac cavities, and the mode of propagation of any murmur which may be present. In this way we shall very frequently be enabled to detect the stenotic condition of the mitral valve even when no murmur at all is present, or when the only murmur audible is one of regurgitation. And this branch of the inquiry is of all the greater importance that it is on the data obtained from it that our prognosis must in every case be founded; the mere diagnosis of mitral stenosis, so readily made in some cases, affording but one, and that in many respects the least important, element in such an inquiry.

Among the most remarkable of these subsidiary phenomena we must reckon irregularity of rhythm, which, always present,\* I believe, in a greater or less degree, becomes so marked in some cases as to constitute a diagnostic phenomenon of considerable importance. This great irregularity undoubtedly depends upon defective nutrition of the cardiac muscle and ganglia; it is not due to dilatation of the left ventricle, which in pure mitral constriction is comparatively rare and never excessive; it is often just as little marked in cases of great dilatation of the right ventricle, as when we have the left ventricle, hypertrophied from secondary causes, acting fairly well to the last, and the right ventricle comparatively unaffected. On the other hand, general debility seems to have considerable effect in developing it, and it is always

\* I speak here only of those who come to us for advice, not of those in whom the lesion is dynamically compensated, nor of those in whom compensation has been restored by treatment, *vide* note p. 123.

lessened by means directed towards improving the general health and especially the cardiac power. But it is chiefly the coexistence of pyrexia which produces its most striking development, and that quite independent of the amount of stenosis present; and it occurs in other forms of cardiac disease under similar circumstances, though never in so marked a degree; its true cause, therefore, would seem to be some lesion of innervation resulting from impaired nutrition of the cardiac ganglia.\* Of course, in mitral stenosis the nutrition of the heart is necessarily always below par, this organ is therefore in this disease specially obnoxious to lesions of innervation affecting motility, whether of reflex or pyrexial origin. The following diagram (fig. 15) graphically represents this extreme irregularity in a sufficiently marked manner.

Fig. 15.



CASE XII.—The patient William Donald, aged 37, admitted into Ward V. on 10th March 1871, was a labourer recently working in a gaswork where he was much exposed to sudden alternations of heat and cold. He had always enjoyed good health till four weeks ago, when he caught a severe cold;

\* The reserve energy of the heart (*vide* p. 34), upon which its recuperative power when exposed to injury or disease depends, seems to result from the fact that the ganglia work well within themselves, and are able to exercise their function for hours (Panum) or many minutes (Von Bezold) after they are completely deprived of arterial blood. Long continuance of imperfect nutrition has, however, a most important influence in modifying the action of the ganglia (Von Bezold, *loc. cit.* s. 279), and as irregular action seems to depend upon an interference of the opposed actions of the inhibiting and accelerating nerves (Bowditch, *Ludwigs arbeiten* 1873; Von Bezold, *Innervation des Herzens*, 1863, S. 297, &c.), there can be little doubt that the modifying influence of malnutrition has a most important effect in producing the obstinate irregularity in the later stages of mitral stenosis, of atheroma of the coronaries, and even of the greatly dilated heart.



since then he had been feverish and ill, with gradually-increasing debility, some cough, and bloody expectoration. On admission he looked somewhat exhausted, his breathing was hurried, his pulse 120 and extremely irregular. On percussion the cardiac dullness was found to be normal, the apex beat being in the usual position. On auscultation over the mitral area, the first sound was found to be of slightly thumping character, impure, but unaccompanied by any bruit; no thrill was perceptible over the apex beat. Between the second and third ribs on the left side close to the sternum, the second sound of the pulmonary artery was heard greatly accentuated. Between the second and third ribs on the right side close to the sternum, the aortic second sound was found to be weakened. At the base generally, but most distinctly about mid sternum on a level with the fourth rib, marked reduplication of the second sound was to be heard. On percussing over the left lung anteriorly, the upper portion was found to be natural, and the lower lobe somewhat dull; posteriorly there was a small dull patch about the centre of the scapular space, from the lower border of the scapular space the lung was dull. On the right side posteriorly there was dullness at the lower border of the scapular space, otherwise percussion over the right lung was normal; over the dull portions respiration was more or less obscured; over the other parts vesicular, mingled with occasional coarse crepitating rattles. The expectoration was catarrhal, largely mixed with pure blood.

The diagnosis in this case was bronchial catarrh, occurring in a person affected with mitral stenosis, and culminating in pulmonary apoplexy (embolic pneumonia).

The history of an ordinary febrile attack, accompanied by cough, catarrhal expectoration, and occasional coarse crepitation over the chest, persisting four weeks after seizure, sufficiently confirmed the first part of this diagnosis. The copious expectoration of blood, coupled with the existence of dull patches, over which the respiration was obscured, also



confirmed the latter part of it.\* While I based the coexistence of mitral stenosis upon—1st, the slightly thumping character of the impure first sound; 2d, the extreme irregularity of the pulse, which is so marked in no other form of cardiac disease that I am acquainted with; 3d, the reduplication of the second sound, which, though it is found under other circumstances, is never so persistent as in mitral stenosis; and 4th, the weakened character of the aortic, and the strongly accentuated character of the pulmonary second sound. The first of these phenomena duly recognised by a practised ear is, I believe, quite pathognomonic of mitral stenosis, being simply the last portion of the murmur still extant, the “b” of the murmur as vocalised, *rrrrb*—all the R’s being expunged. It is almost invariably associated with more or less irregularity of the pulse, though that is by no means always so extreme as it was in the present case. The persistence of the third phenomenon described aided greatly, when coupled with the two former, in confirming the diagnosis, and this was further strengthened by the subsidiary, and, from a diagnostic point of view, comparatively unimportant phenomena of pulmonary accentuation and hæmoptysis.

After admission, the patient gradually fell into a state of low muttering delirium, from which he was roused by the moderate exhibition of stimulants and the free use of digitalis. For a couple of days subsequently he seemed to rally, and faint hopes of ultimate recovery were beginning to be entertained, when symptoms of subacute peritonitis set in, and in two days afterwards he died, on the 20th March, ten days after admission, the cardiac symptoms remaining unaltered to the close. At the dissection, the small intestines were found matted together by recent lymph, they were congested and friable, and the omentum was adherent to their surface, three

\* The occlusion of the bronchi passing through the hæmorrhagic masses, preventing the development of bronchial breathing in such cases, thus in so far confirming Skoda’s theory of the mode of origin of this phenomenon, that of resonance, in opposition to the theory of improved conduction on account of solidification.

murmurs continued as just described, till he was discharged relieved, and declaring himself fit for work.

These cases exhibit in a marked manner the extreme importance of great irregularity of the pulse and cardiac action as a symptom of mitral stenosis, especially when associated with hæmoptysis. Though extremely suspicious under all circumstances, these symptoms may, I believe, be regarded as quite pathognomonic when accompanied by a tolerably persistent reduplication of the second sound, especially if associated with presystolic thrill, notwithstanding the absence of all murmur. The presence of pulmonary accentuation is a phenomenon of comparatively slight importance in specialising the diagnosis, as it is usually absent and rarely well marked when reduplication is present, and is really due solely to pulmonic congestion, which may be the result of many various lesions.

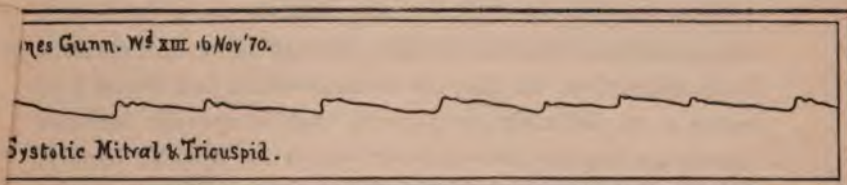
Apart, however, from any presystolic murmur, or from any extreme irregularity of the pulse, mitral stenosis is frequently attended solely by a systolic mitral murmur, and it is not only a matter of interest, but sometimes of great importance, to determine the exact nature of the lesion on which this murmur depends. The two following cases, besides the great individual interest which they possess, are of consequence as showing some of the data upon which this diagnosis may be founded.

CASE XIV.—Agnes Gunn, aged 18, admitted into Ward XIII. on 27th October 1870, complaining of dyspnœa, cough, and spit. There is no history of rheumatism, but she had suffered from chorea about two years ago. About twelve months ago she was admitted with symptoms similar to the present into Ward XI. under Dr Laycock, and in the record of her case it is stated that there was then a mitral murmur succeeding and partially replacing the first sound, and that the tricuspid dulness was increased. At the date of admission under my care, her face was observed to be full, puffy, and rather livid, her expression dull and heavy, yet not free from anxiety. There was some swelling of the feet and legs, great dyspnœa, con-



siderable hard cough, and copious watery expectoration. On passing the hand over the lower part of the sternum, the right side of the heart was found to beat with a slow heaving pulsation, the left apex being only faintly felt in its normal position between the fifth and sixth ribs, the impulse between the third and fourth interspaces on the left side being greater than normal. On percussion, the cardiac dulness was found to extend at one inch from the sternum from the upper border of the third rib down to the liver dulness; and on a level with the fourth rib, from one inch to the right of the sternum transversely across for a distance of six inches. On auscultation in the mitral area, the apex beat was found to be accompanied by a slight thump, followed by a bruit which replaced the first sound; the second sound was distinctly audible at the apex; this systolic murmur was lost as we passed to the left, and, about the middle of the infra-axillary space, was

Fig. 16.



replaced by an impure first sound, which continued audible on auscultating round to the base of the scapula. Over the tricuspid area a loud blowing murmur completely replaced the first sound. At the base, between the second and third ribs on the right side, an impure first sound was heard followed by a feeble second; between the second and third ribs on the left side, the first sound was also impure, and followed by a distinctly accentuated second sound. There was no venous pulsation in the neck, but the veins were somewhat small, empty, and obscured by the general turgidity. The pulse was 76, small, weak, and somewhat irregular; its character is graphically represented in the annexed diagram (fig. 16). She continued under treatment, mainly with digitalis and

squill, till the 29th December, when she was discharged, relieved from her more urgent symptoms.

In this case the main elements relied on in proof of the existence of mitral stenosis were—1st, The slight thump preceding the systolic bruit, and constituting, as it were, an imperfect and abortive attempt at a first sound, a phenomenon which is frequently observed in such cases of mitral stenosis as have no presystolic murmur attending them, and which very possibly is in most cases merely the tail of the rough auricular-systolic murmur, which it phonetically exactly represents, but it is too sharp and short for my ear, at least, accurately to time; in other cases it undoubtedly is due to the imperfect closure of the mitral valve, as can be accurately enough ascertained by timing it by the carotid pulse, the thump in such cases being more prolonged and the systolic murmur not infrequently absent. 2d, The fact that the soft systolic murmur was not distinctly propagated past the middle of the infra-axillary space. This is a very common but not an invariable occurrence in mitral stenosis; not that the systolic murmur is altogether lost, but that the remnant of the first sound is so much more distinctly propagated, that it speedily becomes merely an impure first sound, in which the systolic blow is in such cases more or less distinctly to be recognised. In the purely regurgitant murmur this is never the case; occasionally the murmur may become fainter as we pass to the angle of the scapula, but it never assumes any of the elements of a first sound. These two phenomena when present are perfectly distinctive, and are never observed save when mitral obstruction exists. As corroborative phenomena, we had dilatation of the left auricle, as shown by the unusual pulsation above the fourth rib, which was both seen and felt to precede the ventricular pulsation, and the early occurrence and rapid development of dilatation of the right side of the heart, proving the obstacle to the onward flow of the blood, an obstacle still further evinced by the diminished arterial pulse (*vide* upstroke in fig. 16), the weakened sound of the closure of the aortic valves,



and the persistence of the pulmonic accentuation, notwithstanding considerable tricuspid regurgitation, as evinced by the loud systolic tricuspid murmur. This proof derived from the state of the right side is even more strong in cases in which there never has been any coexistent bronchitis, as is occasionally observed.\* And even in this case, though there was evidently great pulmonary congestion and copious expectoration of watery fluid, yet the absence of rhonchi and the presence of crepitation showed that this condition was much more allied to the general turgescient œdema of the body than to true bronchitis; that it was, in fact, more an œdema of the lung than a true catarrhal condition, and therefore all the more valuable a proof of obstructed circulation.

On 27th March 1871, Agnes Gunn was readmitted for an exacerbation of all her symptoms, induced about a month previously by going on a cold day from the wash-tub to an outside well while scantily clothed. Her cough, shortness of breath, watery expectoration, and cardiac distress were much increased; the jugular veins, though still small, were now seen to pulsate distinctly synchronously with the heart, and her face, neck, and limbs, but especially her lower ones, were very œdematous. The condition of her cardiac organ was much as formerly, with this exception, that now the apex beat was very distinctly to be felt, and the result of this was that the impulse at the lower part of the sternum was apparently lessened. This was explained to be probably due to increased hypertrophy and dilatation of the right ventricle, which had pushed the left wholly backwards, the right apex being now the only one to be felt. It is obvious that this was the only explanation admissible in the face of an increasing cardiac affection, now fast becoming serious, and so largely implicating the right heart from the first; it was impossible to

\* Especially in Ellen Harkins, admitted into Ward XIII. on 20th April, and discharged improved on 17th June 1871. In her we had all the percussion signs of tricuspid dilatation with distinct jugular pulsation, but no tricuspid bruit, and an entire absence of any history of bronchitis.

conclude that a left ventricle, defectively nourished and unable to hold its own from the first, could have recovered itself sufficiently to re-assert its position apart from any well-marked evidence of improvement in the onward circulation, which was entirely absent, while the view taken was perfectly consistent with the ordinary progress of such cases, though the change effected is only rarely observed in such an extreme degree. Gunn remained in the ward till her death, on the morning of 12th July. During this period she had an attack of hemichorea, the sufferings induced by it being rapidly subdued by hydrate of chloral and full doses of arsenic; but it persisted for many weeks as a slight twitching of the thumb and forefinger of the right hand, finally disappearing, however, many weeks before death. This choreic attack rapidly broke down her cardiac power, and she never rallied, dying under symptoms of pulmonary apoplexy, with gradually increasing cardiac asthenia. The autopsy was made on 13th July, thirty hours after death. Her body was moderately fat, her legs very œdematous, her face and lips livid. On opening the thorax, the pericardium was found to be distended by from 15 to 20 ounces of very slightly turbid serum. The heart was considerably enlarged, weighing  $16\frac{1}{2}$  oz.; the right ventricle concealed the left, and formed the apex of the heart; it was much dilated, and its walls hypertrophied, measuring half an inch in thickness; the tricuspid valve was slightly dilated, admitting five fingers easily; on its right cusp, near the free margin, there was a small vegetation about the size of a millet seed, soft and elastic; the right auricle was greatly dilated, not hypertrophied; the aortic valves were competent; the left ventricle slightly hypertrophied, not dilated; the opening of the mitral valve was so extremely contracted as only to admit the point of the little finger; it was very much thickened, and its cusps glued together by their margins; on the auricular surface of each cusp there was a row of small vegetations like millet seeds; the left auricle was slightly dilated, and its walls much hypertrophied, being about twice their natural thickness. The

left lung contained two large and recent hæmorrhagic clots presenting on section a dark venous surface; one of these was situate along the anterior margin of the superior lobe, extending from the apex downwards for a distance of four inches, while it measured transversely one inch and a half; the other was situate in the inferior lobe, near its anterior margin, and was about the size of a large orange; its circumference was pretty sharply defined. The right lung contained no recent extravasations, but in the inferior lobe, immediately beneath the middle of its external surface, there was a yellowish-grey patch of a wedge shape, with its base to the pleura, evidently the remains of an old embolic infarction; over the surface of this lung, and especially at its anterior margins, there were some emphysematous patches. The liver weighed 4 lbs. 2 oz., was much congested, and slightly cirrhotic, the lobules being very distinctly differentiated by the interlobular cellular tissue. The spleen weighed 5 oz., was of firm texture, and contained two hæmorrhagic infarctions, each about the size of a walnut, tough in texture, and of a bright yellow colour. The kidneys were healthy, and weighed each  $5\frac{1}{2}$  oz. The dissection in this case, therefore, completely confirmed the diagnosis during life, while the marked hypertrophy of the left auricle, in contradistinction to the simply dilated condition of the right auricle, in spite of the great preponderance of regurgitation on the right side, is not only a strong proof of the influence of obstruction in originating hypertrophy, but is also a curious fact from a diagnostic point of view, when we consider the entire absence of any presystolic murmur in this case.

The next case which I shall quote is also very instructive as to the signs of stenosis where only the murmur of regurgitation is present.

CASE. XV.—Alex. Milne, a printer, aged 17, admitted into Ward V. on 28th November 1870, complaining of palpitation, cough, shortness of breath, and pain in the cardiac region. His history was, that similar cardiac symptoms had troubled him from the age of six, and that to these



a cough had been superadded about a year ago; since this time the pain in the cardiac region had been much worse; the patient was pale and anæmic-looking, he has never had rheumatism, his family history was unimportant. On inspection, greater pulsation than natural was visible over the cardiac region, especially at its lower part; the apex beat was distinct between the fifth and sixth ribs,  $2\frac{1}{2}$  inches from the left edge of the sternum; on percussing downwards one inch from the left edge of the sternum, cardiac dulness was found to commence at the upper edge of the third rib, and extended down to the liver dulness; on a level with the fourth rib the transverse dulness did not commence till the left edge of the sternum, but extended to the left for a distance of fully three inches; on auscultating over the mitral area, the first portion of a first sound was heard, followed by a loud systolic bruit, and closed by an imperfectly-heard second sound; this systolic murmur was propagated to the right, and was heard distinctly over every part of the right ventricle, followed by a reduplicated second sound, but was not so distinctly propagated to the left, becoming very faint after passing the centre of the infra-axillary space; over the right ventricle a slight but distinct heaving was conveyed to the ear by the stethoscope; between the second and third ribs, on the left side, distinct accentuation of the pulmonary portion of the second sound was audible, the second sound being here reduplicate, as it was also heard to be between the second and third ribs on the right side, where the aortic portion was heard tolerably natural. Over the jugular veins a hæmic murmur was audible, but no pulsation was visible. The pulmonary physical signs were normal, and the cough left him entirely after a short residence in the Infirmary. His digestive system was normal, except that he had little appetite for food. The urine was smoky, contained numerous blood-corpuscles, a few small granular casts, and one-sixth of albumen.

The peculiar character of the first sound, the mode in which



the systolic murmur was propagated, and the persistent reduplication of the second sound, gave me reason to state that in this case also we had to do with mitral constriction; while the tricuspid bruit and evident dilatation, with hypertrophy of the right side, gave a seriousness to the prognosis, which was not lessened by the persistent hæmaturia and the anæmic condition of the patient.

Under treatment his cough speedily ceased, his cardiac symptoms moderated, the pain ceasing, and even his kidney symptoms were modified, the tube-casts being no longer to be found, and the hæmaturia lessened, while his general health was much improved. He was sent to the Convalescent Hospital on 23d February. After being some time at home, Milne became gradually worse, weaker, and more distressed by his cardiac symptoms, and he was in this state readmitted into Ward V. on 6th April 1871. His cardiac symptoms were unaltered, but his debility was greatly increased. His hæmaturia continued, and there was a good deal of general œdema. He was very shortly afterwards confined to bed, suffering much from orthopnœa; and after lingering on in this state he died exhausted on the 24th of May, having spat a good deal of blood during the last few days of his life. At the autopsy there was found great general œdema of the body; the face, which had been livid before death, had become of a roseate hue; a quantity of froth exuded from the mouth; over both lungs there were numerous pleuritic adhesions; the left lung was congested and œdematous; the inferior lobe of the right lung was hepatised, the middle lobe partially consolidated, and the base of the upper lobe also partially consolidated and wholly œdematous. The heart weighed 22 oz. The pericardium was universally adherent—the adhesions tough and fibrous; the right side was dilated and filled with dark clot, the tricuspid opening admitting five and a half fingers; the left side was excentrically hypertrophied and filled with a tough and partially decolorised clot; the mitral opening was constricted, admitting only one finger and a half, its cusps

adherent and greatly thickened ; the aortic valves were competent. The nutmeg liver weighed 3 lbs. 9 oz., the spleen weighed 12 oz., the kidneys weighed each  $6\frac{1}{2}$  oz. ; they were enlarged, the capsules non-adherent, the surface a marble-grey, studded with stellate vessels and hæmorrhagic spots, cortical portion speckled with yellowish opacities.

These cases are sufficient to show the peculiar character of the ventricular-systolic murmur, which certainly indicates the dependence of this form of murmur upon a stenotic condition of the mitral valve quite apart from the existence of any auricular-systolic or of any auricular-diastolic murmur, both of which are frequently entirely absent in such cases. But, while these cases completely disprove the idea that actual mitral regurgitation is never present unless we can carry the murmur distinctly round to the inferior angle of the left scapula, it would be equally erroneous to suppose that when we can carry the murmur of regurgitation right round to the inferior angle of the scapula, unpreceded by any thump and unmodified by any attempt at a first sound, we have to do with regurgitation solely, and not also with stenosis. Among many instances to the contrary I select the following as not only a well-marked example of this, but also as a very instructive instance of the changes which the murmur of mitral stenosis may undergo during the progress of the disease.

CASE XVI.—Andrew Ormiston, a miner, aged 17, admitted into Ward V. on 1st November 1869, complaining of shortness of breath and pain in the cardiac region. Patient states that he has been ailing for about a year, but before that was always in good health, with the exception of having had chickenpox and scarlatina in childhood—in particular, has never suffered from rheumatism. He has been in hospital once during the past year for similar complaints. He is troubled with shortness of breath, aggravated by exertion or coughing ; his expectoration is greyish, but sometimes bloody. He has occasional paroxysms of pain in the epigastrium, coming on about half an hour after taking food. He is small



for his years, but of a ruddy countenance and fresh healthy appearance. He is [pigeon-breasted, the chest being flattened laterally; on palpation a thrill is felt over the heart's apex preceding its impulse. Cardiac dulness extends from the upper edge of the third rib to the liver dulness, and transversely along the upper border of the fourth rib from the right edge of the sternum to the nipple, a distance of four and a half inches. On auscultating over the mitral area a well-marked rough bruit is heard preceding and running up to the first sound; the pulmonary second sound is accentuated, the aortic somewhat weakened. The accentuation of the pulmonary sound is markedly increased whenever he happens to have a cough, and also after exertion. Pulse 88, soft, and only slightly irregular. Respiratory system normal. Tongue clean, appetite variable, bowels regular, urine normal. During his residence in hospital his general symptoms varied according as he suffered from catarrhal complication or not, or from temporary dyspepsia. He was sent improved to the Convalescent Hospital on 15th December. He was recommended to give up mining, and if possible to procure some easy indoor occupation. For some time he took charge of a library; latterly he assisted in a grocer's shop. He grew considerably, and became a well-grown, healthy-looking lad of his years, though quite unfit for the ordinary exertion of manual labour, his sufferings being always increased by any unusual exertion. He came occasionally under observation, without any material alteration of his symptoms or condition, until the 29th of May 1873, when he again came under treatment, with his condition in every way materially deteriorated. His history was that in the preceding February he had a bad bronchitic attack which confined him to bed for three months, and seriously aggravated all his symptoms. So soon as he was able he came to Ward V., when he was found to labour under considerable dyspnoea, aggravated by the slightest exertion, occasional palpitation, and slight cough. But his heart was found to have undergone a most serious alteration

for the worse. The jugular veins could be distinctly seen to pulsate; there was great and manifest heaving at the lower part of the sternum, and very evident pulsation above the fourth rib, and to the left of the sternum, preceding the ventricular pulsation. The breadth of the cardiac dulness at the level of the fourth rib was very much increased, and measured no less than six inches across, extending to the right of the mid-sternum for two inches and one quarter. There was still some thrill to be felt in the mitral area, that is, over the diffused apex beat, the point of strongest impulse being between the fifth and sixth ribs, three inches and one quarter from mid-sternum. In this position there was no longer any presystolic murmur to be heard, but we had instead a loud systolic murmur, propagated distinctly round to the lower angle of the scapula, as well as less distinctly into both the aortic and pulmonary areas. In the mitral area no second sound was to be heard. In the tricuspid area a very loud systolic murmur of a somewhat higher pitch was audible, and here also a distinct second sound was heard. In the aortic area the second sound was much weakened; in the pulmonary area the second sound was reduplicated and also accentuated. Under appropriate treatment, of which digitalis in the form of tincture was the most important part, he gradually improved, his heart closing up, so that even after only a few weeks' treatment the tricuspid murmur was replaced by a first sound, and the mitral murmur was preceded in the mitral area by a portion of the first sound, which at the middle of the infra-axillary space completely took its place. But his feeble constitution had received too severe a shock ever fairly to rally, and the cardiac muscle never fully regained its former condition. The closure of the ventricles was ever after this interfered with by the slightest illness or fatigue, even the ordinary life-work of the day was often too much for it, so that the almost normal tricuspid first sound heard in the morning was often replaced in the evening by a distinct systolic bruit. The presystolic murmur formerly audible never returned. He was



discharged to the Convalescent Hospital on 14th August 1873, considerably improved. But after this he continued feeble, and frequently came under treatment for cardiac symptoms or pulmonary complications, and for the last time on the 4th of September 1874. His cardiac physical signs were very much as last described, a loud systolic bruit, free from all trace of presystolic thump or of the first sound, was heard in both the mitral and tricuspid areas, and was carried right round to the lower angle of the scapula, being also propagated into both the aortic and pulmonary areas, and this underwent no change up to the period of his death, which took place upon October 5th 1874. At the autopsy the body was found to be well nourished, there was œdema of the lower limbs, 6 or 8 oz. of fluid in pericardium, and some effusion into both pleura.

*Heart* much enlarged, weighing 17 oz., all its chambers evidently dilated. Right ventricle especially dilated and elongated, so that its apex projected beyond the apex of the left ventricle, and formed the true apex of the heart. The cavity of the left ventricle was found slightly dilated, and its walls very much hypertrophied. The mitral valve was extensively diseased, its orifice admitting only the point of the little finger; its cusps were greatly thickened and adherent at their edges; each thickened cusp contained calcareous matter. The left auricle was greatly dilated, and somewhat hypertrophied. The aorta was below its natural size, its lumen being only half an inch in diameter, the aortic valves competent and natural. The right ventricle was greatly hypertrophied and dilated; the pulmonary artery was also greatly dilated, its lumen measuring one inch in diameter, but the valves were competent. The tricuspid orifice was dilated, capable of admitting seven fingers, the right auricle greatly dilated, but not hypertrophied.

*Lungs.*—The right, weighing 1 lb. 15½ oz., contained several masses of circumscribed effused blood, some quite dark and recent, others pale and grey; the whole of the middle lobe was rendered solid, the lung was free from pleuritic adhesions. The left lung was bound by firm adhesions to the chest, it

was engorged and œdematous; the base was solidified as if from old pulmonary apoplexy.

*Liver* was intensely congested with venous blood, and somewhat cirrhotic; it seemed to be somewhat fatty; it weighed 3 lbs. 10½ oz.

*Spleen* weighed 6 oz.; hard, fibrous, and congested.

*Kidneys* (5½ oz. each) somewhat congested.

It would be difficult indeed to find a more instructive case than this, where we have five years of intelligent observation of a most interesting cardiac lesion, followed by a confirmatory *post-mortem* history. This case is indeed an epitome of all that is most interesting in the history of mitral stenosis, with this sole, and in so far remarkable, exception, that the murmurs, though they varied, did not vary capriciously, nor were they at any time much altered by position, though so long as any trace of a presystolic thump could be perceived this was much increased by the upright position and by exercise; when however this presystolic thump had finally disappeared, it was not restored either by exercise or position, the case then presenting a well-marked example of all the phenomena of what I may term incurable mitral regurgitation—a regurgitation depending upon actual disease of the mitral valve in contra-distinction to curable mitral regurgitation, a regurgitation depending on simple muscular dilatation apart from any actual disease of the mitral valve—the phenomena of which I shall presently describe. In all its stages there was well-marked pulsation above the fourth rib to the left of the sternum, which could both be seen and felt distinctly to precede the pulsation below the fourth rib and the apex beat; this superior pulsation was therefore, both in position and rhythm, coincident with the left auricle and its systole, just as the inferior pulsation was coincident with the left ventricle and its systole. This auricular pulsation was, as it always is in such cases, synchronous with the apex thrill and the presystolic murmur, showing the dependence of these phenomena upon the systole of the auricles,



and not upon that of the ventricles, which they distinctly preceded.

The next point of interest in this case is that the presystolic murmur persisted, *per se*, so long as the cardiac muscle retained its normal tonicity, while so soon as the resulting pulmonary congestion became sufficiently great to determine loss of tone and secondary dilatation of the right ventricle, we had the presystolic murmur gradually decreasing to a mere presystolic thump preceding the mitral systolic murmur, an impure first sound becoming in this stage audible about the middle of the infra-axillary space as the stethoscope was moved to the left in the plane of the apex beat. Whenever the weakness of the cardiac muscle became extreme, as it did during the few last weeks of this patient's life, even this thump disappeared, a pure systolic murmur alone remaining, differing somewhat in pitch over the tricuspid and over the mitral areas, yet capable of being carried round from the mitral area to the lower angle of the scapula without any break or alteration of its pitch. This systolic murmur was not solely due to tricuspid regurgitation,\* because appropriate treatment at first restored the tricuspid first sound more or less perfectly, but did not also restore the mitral first sound, never doing more than merely bring back the presystolic thump preceding the mitral systolic murmur, as well as an impure first sound to the middle of the infra-axillary space. In other cases we are more successful. A patient comes under treatment with a systolic murmur over the left apex, or it may be over both apices, and by appropriate treatment we first bring back a first sound over the right apex with a thump preceding the mitral systolic murmur, and by and by, as the

\* In the case of John M'Owen, first admitted to Ward IV. on Dec. 10, 1878, and since then repeatedly under observation, I have satisfied myself that this is true, and that though it seems a plausible idea to refer the systolic murmur solely to the right side, and its progress to the left to the gradual dilatation of the right ventricle, this is not really the case. M'Owen had a very remarkable pigeon-breast, and his left apex never left the chest wall, yet after a time he had only a systolic murmur which could be carried right round to the back.

cardiac muscle regains strength, we have a distinct presystolic murmur developed,\* followed by a mitral systolic murmur, or in still more favourable cases by the usual accentuated first sound. In the case before us we had only temporary and very imperfect attempts at closure of the right ventricle, and never any reproduction of the presystolic murmur after it had once disappeared, and it seems to be a matter of some importance to inquire why it is that in certain cases of mitral stenosis it is more easy to produce comparative rehabilitation of the cardiac muscle than in others. The answer to this is, I believe, to be found in the degree of stenosis present, but especially in the period of life at which stenosis has primarily occurred.

We can easily understand that when blood is detained in the lungs by the stenotic condition of the mitral valves, the intra-pulmonary blood-pressure rises with the gradually increasing congestion. So long as the cardiac muscle retains its normal tonicity, the right ventricle for long successfully resists the dilating force thus applied to its interior, even though the intra-vascular pressure within the lungs be sufficient to produce, as in this case, considerable dilatation of the pulmonary artery, which probably commenced long before the giving way of the tricuspid valve. The right auricle, it is true, very early becomes affected, and consequently there are but few cases of mitral stenosis in which increased breadth, to the right of the sternum, in the auricular region—the plane of the fourth rib—may not be detected. But so long as the tricuspid valve acts normally, this distention of the right auricle interferes but little with the carrying on of the circulation, and such cases may persist for years, presenting few, if any, symptoms of the serious disease with which they are affected. But no sooner does the cardiac muscle lose its tone than all this is changed, and serious cardiac symptoms at once set in. In this climate this loss of

\* In Case I., p. 48, will be found an instance in which a presystolic murmur became developed as the cardiac muscle regained strength.



tone is most frequently brought about by an attack of bronchitis or feverish catarrh,\* and we constantly find patients, whose mitral stenosis was either congenital or dated from early infancy, ascribing their illness to a bronchitic attack which had occurred only a few years previously. The loss of vital tonicity in the cardiac muscle of necessity involves a yielding of the cardiac walls to internal pressure, often, as we shall presently see, when that pressure is only normal or but little more,† and of course this happens all the more readily and all the more completely the more abnormally excessive the intra-vascular pressure is, even in the normal condition of the heart. For this reason, a severe attack of bronchitis is not infrequently accompanied by tricuspid regurgitation. And when bronchitis occurs in a lung or lungs already congested by the obstruction to the onward flow of the blood presented by stenosis of the mitral valve, and the muscle of the right ventricle, already slowly yielding before the abnormally increased intra-vascular pressure starting from that stenotic valve, experiences a great and sudden loss of tonicity due to febrile relaxation and malnutrition, the result cannot be otherwise. But dilatation of the right ventricle and the regurgitation through the tricuspid valve which then occurs, are changes full of ominous import to the safety of the patient. Hitherto he has been gliding along the calm though treacherous stillness of a mute disease, now he is plunged over the brink into the midst of those rapids where, with but few quiet intervals, he must for the future do battle for his life.

Whenever mitral stenosis is formed the blood gradually though slowly accumulates behind the stenotic valve, and it is to this slow transference of the blood from the arteries to the veins, accompanied by an equally gradual impairment of the nutrition, and consequently of the action of the cardiac muscle, that death from asthenia, accompanied and indicated by the gradual accumulation of serum in the cavities of the body, is

\* *Vide* Lectures VI. and VII.

† *Vide* Lecture VI.

ultimately due even in the most favourable cases. Though, of course, it is only in the rarest instances—if ever—that this natural result of mitral stenosis is not precipitated by some such accident as that to which I have just referred. The greater the stenosis present, the more rapidly—*cæteris paribus*—this result is necessarily attained, and the more readily of course it may be precipitated. We know that a dilated right ventricle, the result of the increased blood-pressure due to remediable congestion of the lungs in simple bronchitis, the heart being otherwise normal, is readily recovered from. We also know that any persistent cause of increased blood-pressure in the lungs, such for instance as is produced by the limitation of the area of the pulmonary capillaries by destruction of the air-cells in emphysema, induces a permanent and incurable dilatation of the right ventricle. And we can readily understand that any permanent cause of pulmonary congestion, such as we have in mitral stenosis, must have a precisely similar effect in preventing rehabilitation of the dilated right ventricle just in proportion to the amount of stenosis present. But in many cases of extreme mitral stenosis we have to do with another alteration of the vascular system, which not only increases the rapidity with which the blood accumulates in the veins, and the readiness with which compensation may be ruptured, but which also indicates that this stenosis has occurred at an early period of life. Thus we come to regard the occurrence of mitral stenosis in early life as more serious than when it comes on at a more advanced period, and the earlier in life we find stenosis existing, the more grave our prognosis ought to be, even when we can definitely connect it with some preceding rheumatic attack.

As we shall afterwards see, the first effect of a rheumatic attack is pyrexial relaxation of the cardiac muscle and the production of a murmur of regurgitation, frequently audible in the mitral area, more often in the auricular area,\* but of a curable character, which disappears naturally or under treat-

\* *Vide* Lecture VI.



ment at the close of the attack. But we may also have an endocarditis of an acute, sub-acute, or chronic character, of rheumatic origin, which shrivels, thickens, and often mats together the fibrous tissues of the mitral valves and of the *chordæ tendineæ*; this may start simultaneously with the muscular relaxation, or may follow it more or less slowly, with or without an intervening period of apparent cardiac health. The inevitable termination of this endocarditic attack is more or less constriction of the mitral opening. As the cardiac tissues may thus be affected by rheumatism in a two-fold manner, so we may or may not have first of all a curable form of mitral regurgitation; and second, with or without this curable form of mitral regurgitation preceding it, we may have an incurable form of mitral disease developed, the characteristic symptoms and signs of which are those of mitral constriction, which take about a year fully to mature. When this result of rheumatism occurs in adult life, this constriction follows the usual course, and has attached to it the ordinary prognosis of mitral stenosis. But should a rheumatic attack in childhood or early youth be followed by any considerable mitral stenosis, the effect of this must be to accumulate blood behind the stenotic valve, to diminish the wave of blood sent forward, and thus to lessen the normal distention of the aorta and to impair the nutrition of all the tissues. In this way there is produced that arrested development so well known as the constant result of serious cardiac disease occurring in early life, technically termed hypoplasia, affecting primarily the aortic arterial system, and secondarily all the organs of the body. Of this we had a moderate example in this patient (Ormiston), in whom the aorta measured only half an inch in diameter. But this hypoplasia is occasionally much greater; Allan Burns relates one case in which the aorta barely admitted the little finger.\* Such an extreme degree of hypoplasia of the aortic system points, of course, to the development of

\* *Observations on some of the most Frequent and Important Diseases of the Heart.* By Allan Burns, Edinburgh, 1809, p. 32.

mitral constriction at a very early period of life, it may be even to a congenital stenosis of this valve, as Allan Burns has supposed;\* and the existence in some cases of an extreme amount of mitral stenosis with a high degree of hypoplasia of the aortic system, and of course of every organ of the body, quite apart from any trace of rheumatism or of any known efficient cause of endocarditis, certainly favours this supposition.† Be this as it may, however, the important point for us to consider is, that this hypoplastic condition of the aorta, however produced, increases very considerably the difficulty of rehabilitating the right ventricle, once it has become dilated, presenting as it does a second and most material obstacle to the onward progress of the blood, which strengthens and reinforces that already depending upon the stenotic condition of the mitral valve. Hence there can be no doubt, that the earlier the period of life to which mitral stenosis can be traced, so much the more readily is dilatation of the right ventricle and tricuspid regurgitation produced by even comparatively slight secondary causes, and so much the more serious must be our prognosis, because under such circumstances it is never possible perfectly to rehabilitate the right ventricle, and the necessarily fatal transference of the blood-pressure from the arteries to the veins is inevitably hastened.

You will remember then that mitral stenosis is frequently

\* His sixth species of congenital malformation of the heart was constituted by these cases where the mitral valve is malformed, leaving merely a small opening leading from the left auricle into the ventricle.—*Op. cit.* p. 12.

† Virchow, in his pamphlet "*Ueber die Chlorose und die damit zusammenhängenden Anomalien im Gefässapparate*," Berlin, 1872, pp. 18, 19, attributes the valvular endocarditis affecting chiefly the mitral but occasionally also the aortic valves in cases of hypoplasia to the irritating results of intravascular pressure, the ventricle having more blood to send forward than the aorta can receive. This would, however, of itself be much more likely to produce dilatation—as it not infrequently does—than endocarditis. Besides, congenital hypoplasia of all the organs is extremely unlikely to produce any formation of blood in excess of the requirement or capacities of the organism. On the other hand, intra-uterine or infantile endocarditis, causing stenosis of the mitral valve, must inevitably lead to hypoplasia of the aorta and of all the organs to which it is distributed, by cutting off the supply of nutriment during the period of development.



the result of rheumatism, that some degree of it is invariably present whenever the mitral valves have become diseased as the result of endocarditis, however that may have been produced, whether by rheumatism or by any other of those many causes of endocarditis which our present ignorance only permits us to slump together as of non-rheumatic origin, but which are of frequent occurrence. Whenever this stenosis occurs in infancy or early childhood, some degree of aortic hypoplasia is probably always concomitant. And lastly, there is every reason to believe that mitral stenosis is by no means infrequently congenital, though as yet we know not whether as a simple malformation, the result of imperfect development, or of intra-uterine endocarditis, as is most probably the case, and in these instances it is invariably associated with hypoplasia of the aortic arterial system, and consequently of the body generally. Further, you will also remember that this stenotic condition of the mitral valve is to be recognised by the existence of a rough murmur preceding and running up to the apex beat and the carotid pulse, which is pathognomonic of this lesion. This murmur may, and often does, commence as a somewhat musical diastolic murmur following the second sound, becoming rougher towards its conclusion, audible sometimes over the whole cardiac area, more usually, *quoad* its diastolic portion, just over the mitral valve where the fourth rib on the left joins the sternum. Any portion of this prolonged murmur may be absent, and it may either be continuous or broken by a pause equivalent as to rhythm, though not as to duration, to the normal soundless interval of the heart's action, the length of the pause being dependent on the amount of stenosis present, *i.e.*, the shorter the pause the greater the stenosis. A diastolic mitral murmur is of course unaccompanied by any signs of aortic regurgitation, it has a clear second sound preceding it, and is accompanied by no jerking thrill of the pulse increased or produced by elevation of the arm, and no dilatation of the left ventricle evinced by depression of the apex beat; it is probably always slightly musical in character, and chiefly

audible in the mitral area or over the valve. A short, soft, blowing diastolic murmur, even though only audible at the sternal end of the fourth left rib, must always be viewed with suspicion as probably of aortic origin. The distinctive signs of pulmonary regurgitation are practically unknown, but the affection is so extremely rare that it may be excluded; moreover, the signs and symptoms of mitral stenosis, though they would not exclude pulmonary regurgitation, would yet be most unlikely to accompany it, their absence, therefore, would be something in favour of pulmonary regurgitation, and though their presence could not disprove it, it would make it unlikely. Extreme irregularity of the pulse with or without pyrexia is indicative of the possible existence of mitral stenosis, and this is corroborated by the existence of a thump preceding or accompanying the apex beat, by hæmoptysis, signs of enlargement of the right side of the heart, and accentuation or reduplication of the second sound, the accentuated portion of the second sound being always that of the pulmonary artery, and most distinctly audible at the third left costal cartilage.

A simple systolic apex bruit—a murmur of mitral regurgitation—is occasionally the sole indication of mitral stenosis, but in these cases we often have it preceded by a more or less evident thump, the bruit ceasing at or about the middle of the infra-axillary space, being there replaced by a more or less impure first sound. If we can carry the systolic murmur of mitral stenosis right round to the back, then we invariably have a heaving impulse at the lower part of the sternum, accompanied by a systolic murmur of a different pitch, indicating considerable dilatation of the right side with tricuspid incompetence, and accompanied by marked hepatic pulsation. In these cases, as well as in all cases of mitral stenosis however revealed, we have very generally more or less pulsation in the auricular area above the fourth rib to the left of the sternum, a pulsation which is usually entirely wanting, and is at all events very inconsiderable, in cases of simple regurgita-



tion apart from stenosis of the mitral opening. In cases of mitral constriction we frequently hear a systolic murmur in this auricular area; but indeed a more or less distinct murmur in this area is one of the earliest indications of mitral regurgitation from whatever cause.

The prognosis of mitral stenosis is comparatively favourable so long as the right side is unaffected, and the rough presystolic murmur persists, the favourable character of this prognosis being subject to modification according to the probable date of origin of the stenosis, being more favourable the later in life this has occurred. But whenever the right ventricle becomes affected with secondary dilatation, the prognosis becomes at once more serious, and all the more serious the earlier in life this takes place. Because the probability is greater that the stenosis dates from early youth, childhood, or infancy, and the earlier the probable date of the stenosis in such cases, so much the more certain is the presumption of hypoplasia of the aortic arterial system. The co-existence of aortic hypoplasia with dilatation of the right ventricle at once renders the prognosis most grave, and reduces the expectation of life from many to only a few years—probably to not over two or three years, except under most favourable and exceptional circumstances. Sufferers from mitral disease are very much less liable to sudden death than those labouring under aortic incompetence, they die normally from asthenia preceded by dropsy. Many pass through a long life with comparatively little inconvenience, most however suffer at times a good deal from breathlessness, palpitation, and irregular action, the latter being often very persistent, especially during the latter years of life. Embolism, especially of the lungs, is a common occurrence, and frequently cuts short such lives, while in all ruptured compensation, arising from various causes, is much more common than in aortic incompetence, but of much less serious import, being readily recovered from except under the exceptional circumstances already referred to. The treatment of such cases is simple enough,



and resolves itself mainly into the employment of tonic doses of the tincture of digitalis, ten to fifteen minims every four hours, or only twice or thrice a day, according to circumstances. Arsenic is useful when cardiac pain is present, and iron as a hæmic tonic when it agrees. Other remedies must be employed with caution *pro re nata*, as most of the concomitant morbid phenomena in such cases group themselves secondarily round the central cardiac lesion upon which they depend. Nutritious diet and abundant rest are imperatively required, and stimulants in moderation are often useful, but must be employed with judgment as required. Further, I may remark, as we never have mitral stenosis without regurgitation, some degree of hypertrophy of the left ventricle is almost always present, but it is of slow development and never excessive; marked dilatation of the left ventricle is, however, less common, and is traceable to pre-existing pyrexia or increased obstruction from secondary causes, such as arterial atherosclerosis or secondary cirrhosis of the liver, kidneys, &c., the result of venous *remora*.

## LECTURE VI.

ON CURABLE MITRAL REGURGITATION; ITS VARIOUS CAUSES, THE PHYSICAL SIGNS BY WHICH IT IS REVEALED, AND ITS TREATMENT.

GENTLEMEN,—In a former lecture\* I pointed out that the only unequivocal proof of the existence of actual disease of the mitral valves, is the presence of an auricular-systolic, or as it is commonly termed, a presystolic murmur, or, at all events, sufficient proof of the occurrence of such a murmur at some former period of the patient's life; for though this murmur frequently disappears or alters its character, the lesion upon which it depends is permanent. My statement would have been more correct had I said that the only unequivocal proof of disease of the mitral valves is the determination of a constricted condition of the auriculo-ventricular opening, however that may be established. For constriction of that opening is the usual condition when the mitral valves are diseased, and though it is always present when a presystolic murmur exists,† it also frequently exists

\* Lecture IV. p. 110.

† I have no hesitation in making this statement, notwithstanding that Professor Flint says, "I have recorded three cases in which this (presystolic) murmur was loud, and the mitral valves were found *post-mortem* normal. In each of these cases there were aortic lesions rendering the aortic valves insufficient."—*Lectures on Diseases of the Heart*, Philadelphia, 1870, p. 207. Obviously Flint had not timed these murmurs correctly, for a murmur depending on aortic regurgitation is always diastolic, and therefore has a rhythm very different from a presystolic murmur. But even without timing these murmurs, they have each of them such a distinctly different character that no one

when that murmur is not only absent at the time of examination, but has never been recognised at any past period of the history of the case, though even in these instances its existence is frequently revealed by certain signs to which I have already referred.\* As in that lecture, however, I referred chiefly to the murmur distinctive of disease of the mitral valve, the statement may be accepted as correct to that extent. At present I intend to refer to mitral regurgitation unaccompanied by disease of the valve, and shall have to traverse cursorily a wide series of diseases in which this lesion is found present more or less frequently, and in a large proportion of which it is happily permanently curable, though revealed by what to many even yet seems the certain sign of an incurable organic cardiac disease—a mitral systolic murmur. The importance of this observation needs no comment. To the patient it involves the issues of life and death, a comparatively short illness, and thereafter a normal and it may be a long life; or permanent ill health, an abnormal life, and shortened days. To the physician it brings home the paramount importance of an accurate diagnosis, and substantiates in a most striking manner the value of appropriate treatment.

Daily experience in the sick-room has long since taught all of us that muscular debility and relaxation are the inevitable results of all febrile diseases; theoretically we know that the heart is a muscular organ, that even in sickness it never rests, and that the influence of malnutrition—unrepaired waste—so evident in the other muscles, must be even still more predominant in it. But how few of us have recognised the different modes in which this malnutrition and relaxation of perfectly acquainted with the presystolic murmur could ever mistake a diastolic aortic murmur for it. Flint's theory that the regurgitant blood floats up the segments of the mitral valve and produces a practical constriction of the opening, is obviously no explanation. In the normal state of the heart "the segments of the valves at the end of the ventricular diastole are so close as to be nearly in contact" (Pettigrew, *Trans. Roy. Soc. Ed.*, vol. xxiii. part 3, p. 796). According to Flint's theory, a presystolic murmur ought to be a constant phenomenon, not only in all cases of aortic incompetence, but also in health.

\* Lecture V. p. 134.



tissue may make itself known. When diminution of the mass of blood accompanies an absolute wasting of the tissues, as in phthisis, the heart simply atrophies in all its dimensions, and its cavities are correspondingly diminished in size (concentric atrophy), and so invariable is this the case, that one eminent author (Walshe) states that the sustainment of the heart's weight at the par of health in those slowly cut off by such exhausting diseases as phthisis or carcinoma must be regarded as the equivalent of hypertrophy in those free from such diseases.\* In the case of other diseases, some of a more distinctly febrile character, such as typhus, enteric, and relapsing fevers, erysipelas, and probably many others, as well as in others of a less febrile type, such as chorea, and some forms of spanæmia, especially chlorosis, the change in the condition of the heart consists not in any tendency to atrophy, but rather to such a complete relaxation of its tissues, that in its extreme form it seems as limp as a piece of wet paper,† yet without any very evident diminution in its bulk, or any marked alteration in the relative dimension of its cavities. This altered condition of the muscularity of the heart is always preceded and accompanied by a more or less evident alteration in the blood. It may be diminished in quantity—anæmia, but this condition is of short duration, and speedily passes into what Beau has called serous polyæmia, a state perhaps better known by the name of spanæmia. And it is, I believe, to the greater predominance of this form of hæmic lesion, as well as to the comparative amount of cardiac relaxation present, that we are to attribute the constant prevalence in some diseases, such as chlorosis, of certain peculiar phenomena which are only occasionally found in other diseases in which muscular relaxation and blood alterations are also present, whether these diseases are febrile or non-febrile in character.

As the cardiac and hæmic phenomena to which I refer are

\* *On Diseases of the Heart*, 3d ed. p. 274.

† Quoted from Louis by Stokes, *Diseases of Heart and Aorta*, Dublin, 1854, p. 368.

most typically developed in chlorosis,\* I shall first of all direct your attention to the character and the explanation of these phenomena as they are found to occur in that disease, and thereafter employ the information thus obtained in the explanation of similar facts occurring in the course of other diseases. And in doing so, I shall not particularly enter into the pathology of chlorosis generally, but simply describe it as a form of spanæmia of common occurrence, especially in country girls who for the first time find themselves exposed to the unhealthy surroundings inseparable from domestic service in a town, and in whom the prominent derangement of the genital functions is almost invariably amenorrhœa. It is also of no infrequent occurrence in town-bred girls of a better class, especially about the time of puberty, and in them the menstrual discharge is pale, usually scanty—menorrhagia being exceptional, and always followed by great exhaustion. In such patients the lips externally may be rosy, and the cheeks present a certain amount of youthful bloom, but the interior of the lips, the gums, and especially the conjunctivæ of the lower eyelids, are pale and bloodless. The face is rather puffy than clear cut or sharp in its outlines; the ankles tend to swell, the appetite is irregular and defective, and there is breathlessness and palpitation on the slightest exertion. This condition is also signalled by the occurrence of what are termed functional or hæmic murmurs, which are of constant occurrence in the veins, forming the well known *bruit de diable*, or humming-top murmur, and are not unfrequently heard in the aortic area, more frequently in the pulmonary area, and more rarely in the mitral area. The venous murmur is continuous and audible both during systole and diastole, its intensity increasing with the thoracic and cardiac aspiration, and diminishing with expiration and the

\* I refer here to the ordinary and curable form of chlorosis commonly met with, and not to that incurable form, accompanied by hypoplasia of the internal organs, referred to by Virchow in his pamphlet, *Ueber die Chlorose und die damit zusammenhängenden Anomalien im Gefässapparate*, &c., Berlin, Hirschwald, 1872. And also by Rokitansky, in his *Handbuch der Pathologischen Anatomie*, Bd. i. s. 158, and Bd. ii. ss. 418 and 585. 1840-44.



cardiac systole, so that the venous hum swells and dies as those alternate. The cardiac murmurs are always systolic in rhythm.

Much of the interest of these murmurs centres, as you may well believe, in their causation. If we regard them as purely hæmic in character, then, however difficult it may be to understand their production, their removal is as simple as we know it to be easily produced, and possessed of no great general pathological importance. But if we can show, as I hope to do, that the cardiac portion of these murmurs is not simply hæmic or purely functional in character, but is the product of an actual cardiac lesion, then we link blood disease with cardiac lesion in a very striking manner, and obtain the important pathological information that some cardiac lesions are certainly and permanently curable.

I need not test your patience nor occupy your time with an account of all the various theories which have been propounded in explanation of the production of venous murmurs. It is enough that the modern science of physics has proved that sound in relation to fluids is always caused by the production of Savart's fluid veins at the point where it is heard.\* Now, venous murmurs are readily produced in spanæmic individuals by moderate pressure over any vein of tolerable size, such as the external jugular. They are also heard over the torcular Herophili, over the upper part of the innominate veins, especially the right one, and over other parts of the venous circulation where there exists naturally what is produced artificially by pressure, a flow of blood through a part which is relatively constricted to that beyond it. Besides relative constriction, however, a certain amount of force is requisite to produce these sonorous veins. Hence they are frequently inaudible in the recumbent position, becoming at once audible when the venous flow is hastened by elevating that part of the body at which we may be listening. It is true, as has been noticed by many observers ("London Heart Committee," Liman, Winterich &c.), that venous murmurs are frequently audible in individuals who are in apparent health; but we are always oscillating

\* Lecture I. p. 43.



between health and disease, and are regarded as healthy so long as we keep within certain well-defined limits. All that this observation proves, therefore, is, that a certain amount of spanæmia is not inconsistent with apparent health; it by no means proves that venous murmurs are compatible with perfect health, or, what is synonymous, with perfect blood. As a venous murmur is often to be heard in certain positions, such as the torcular Herophili, where no other change is at all probable, under all the circumstances of the case, except one involving an altered relation of the blood to its containing vein, we can have no hesitation in ascribing its production to that cause. And, further, from what we know of the mode in which fluid veins are formed, we can have no difficulty in saying that this altered relation of the blood to the walls of the vein must consist in the production of increased friction between the two, so that in those positions where there is normally a relative constriction, insufficient, however, to produce sonorous veins, the increase of friction between the wall of the vein and the layer of blood next it practically narrows the opening sufficiently to do so, by retarding the exterior portion of the blood current and leaving the central or axial portion uninfluenced. Physical laws leave no doubt as to this. It is, for physiologists to explain whether it depends simply upon a watery condition of the blood or upon some other cause, and their explanation would no doubt be of the greatest importance for practical medicine.

But the venous murmurs which accompany chlorosis are for many reasons of very much less interest and importance than those heard in the cardiac area. As already mentioned, these are systolic in character, and on carefully examining all the cardiac areas, we discover that the position of maximum intensity of this so-called arterial bruit is not over any artery at all, nor in any of the usually accepted areas of cardiac sounds and murmurs, but is actually about one inch and a half, or rather more, to the left of the pulmonary area, and in the same plane immediately over the part where the appendix of the left auricle pops up from behind, just to the left of the pulmonary artery.

This so-called arterial murmur is, therefore, not arterial at all, but strictly auricular in its source. This is a most important fact; it at once links this chlorotic murmur with others whose source we distinctly know, and gives us an unmistakable clue to its true point of origin.

We all know very well that when the first sound in the mitral area is replaced by a murmur, this murmur is propagated with diminished intensity into the pulmonary and also into the aortic areas. But it has also been observed that the systolic, basic, and apparently pulmonary murmur accompanying mitral disease is occasionally actually louder than that audible in the mitral area, and that a murmur of mitral regurgitation is sometimes only to be heard in the first mentioned area. Skoda\* was the first to point this out, and he attributed it to a hypothetical alteration of the lining membrane of the pulmonary artery in such cases. Joseph Meyer† has shown that this hypothetical alteration is a myth, and has explained the occurrence of this phenomenon by another equally untenable supposition. But it was reserved for Naunyn‡ to show that this murmur of mitral disease, whether it accompanies a systolic murmur in the mitral area, or is itself the sole indication of mitral regurgitation, has its position of maximum intensity, not in the pulmonary area, but one inch and a half or more to the left of the left edge of the sternum, in the second interspace; that is, as he has shown, and as anatomy teaches us, not over the pulmonary artery at all, but exactly over the spot where the appendix of the left auricle comes up from behind, just to the left of that artery. Naunyn explains this phenomenon by attributing it to the better conduction of the murmur along the course of the regurgitating blood, the fluid veins producing sonorous vibrations louder at the point of impingement than at that of origin; and to the circumstance that the dilated auricle

\* *Abhandlung über Percussion und Auscultation*. Wien, 1844, s. 233.

† *Virchow's Archiv*, Bd. ii. heft 2, s. 277.

‡ "Ueber den grand weshalb hin und wieder das systolische Geräusch bei der mitral Insufficienz am lautesten in der Gegend der Pulmonalklappe zu vernehmen ist"—*Berliner Klinische Wochenschrift*, 1868, No. 17, s. 189.



n all such cases is closer than usual to the anterior surface of the chest, and therefore nearer to the ear.\* This condition of things is not uncommon in mitral constriction, and there are many circumstances which in this state contribute materially to the production of this phenomenon. At present, what I wish to point out is, that in chlorosis, in which all these phenomena, to which I may now comprehensively refer under the head of cardiac dilatation consecutive to spanæmia, exist only in the very slightest degree, this pulmonary or rather auricular murmur is always present, and is often—so often as to constitute it almost invariably—the sole sign of mitral regurgitation in these cases.

As you all very well know, the primary or typical hæmic murmur is basic in position, and unlike most other murmurs found in this position, “it has no definite line of propagation.”† This basic position of the primary hæmic murmur of course excludes Parrot’s‡ theory, of its tricuspid origin; while the absence of propagation along the course of the arteries renders untenable the classic hypothesis of its aortic origin supported by Hope,§ Bellingham,|| Potain,¶ and others, and though Marey\*\* has of late reverted to this idea, apparently because it suited with his fancy that a low blood tension in the aorta

\* This peculiar phenomenon has also been referred to by Bamberger, Friedreich, and others, and explained after different methods. Recent writers have all, however, adopted Naunyn’s explanation—*vide* Paul Niemeyer, *Handbuch der Percussion und Auscultation*, Bd. ii. abtheil i. s. 140, Erlangen, 1870; and Gerhardt’s *Lehrbuch der Auscultation und Percussion*, 2d edition, Tübingen, 1871, s. 283. Curiously enough I find that Von Dusch, whose work was published in the end of 1867, though dated 1868, and therefore preceded Naunyn’s paper, takes a very similar view in regard to the pulmonary systolic murmur heard in such cases. He says, “Ich glaube, dass dasselbe—this murmur—durch die bei insuffizienz der mitralis im linken Vorhofe entstehenden pulsirenden Bewegungen hervorgebracht wird, welche sich der ihm anliegenden Pulmonalarterie mittheilen und die Blutströmung in derselben beeinträchtigen.”—*Lehrbuch der Herzkrankheiten*, Leipzig, 1868, s. 205.

† Hayden, *Diseases of the Heart and Aorta*, Dublin, 1875, p. 252.

‡ *Archives Gén. de Médecine*, 6<sup>me</sup> série, tom. viii. 1866; vol. ii. p. 158.

§ *A Treatise on Diseases of the Heart*, London, 1839, p. 106.

|| *Diseases of the Heart*, Dublin, 1853.

¶ *Dictionnaire Encyclop. des Sciences Médicales*, tom. iv. p. 392.

\*\* *Physiologie Médicale de la circulation du Sang*, Paris, 1863, p. 479.



favoured the formation of fluid veins at its orifice; yet Marey's support is of no value when opposed to contradictory facts, while it is unfortunate for his theory that this murmur is always best marked, and often only to be heard, in the neighbourhood of the pulmonary artery, where a well-marked accentuation of the second sound assures us that the blood tension is actually increased. So usual and well defined, indeed, is this position of maximum intensity, that Marshall Hughes\* has suggested the pulmonary artery itself as the source of this murmur. But this hypothesis is excluded, first of all because no murmur of strictly pulmonary origin could possibly be referred to all the four orifices in turn, as has been the case with the hæmic murmur; and second, because however singular a murmur of regurgitation in this position may seem to be, its causation is by no means difficult to understand.

The spanæmia of chlorosis is what Beau has termed a serous polyæmia, a state analogous to that following repeated venesections in which the red corpuscles are diminished and the serum increased. In this condition there is relaxation of the whole muscular system including the heart, hence there is progressive dilatation of that organ, not due to residual accumulation from defective power of expulsion, but simply to loss of elasticity. This dilatation is primarily compensated by means of the reserve force of the heart, and ultimately by the development of hypertrophy, as already detailed in relation to the similar lesion in aortic incompetence.† Beau, when experimenting upon dogs and rabbits by bleeding them to death, found that the heart of those animals in whom serous polyæmia had been fully developed, was not only larger, more dilated, but also weighed from one-fifth to one-sixth more than that of healthy animals killed by one fatal hæmorrhage from a severed carotid.‡

\* *Guy's Hospital Reports*, second series, vol. vii. p. 161.

† Page 84.

‡ *Archives Generales de Médecine*, 4<sup>ime</sup> série, tom. ix., Paris, 1845, p. 156. The following case proves that a similar condition is not unknown in man. W.R. æt. 55, admitted into Ward XXXII., New Royal Infirmary, April 27th 1880, labouring under serous polyæmia of a serious character (pernicious anæmia)

and as the heart in chlorosis has also been noted as large by Beau\* himself, by Bamberger,† Friedreich,‡ Wanderlich,§ and Stark,|| the most probable assumption is that it is produced in a similar manner. Indeed the cardiac murmurs found in chlorosis are developed in a manner so strictly in accordance with this assumption, as to afford an almost conclusive proof of its truth. For first of all we have the venous hum of serous polyæmia, next we have an accentuated pulmonary second, followed closely if not accompanied by Naunyn's auricular murmur of mitral regurgitation, then a systolic murmur in the mitral area, sometimes a distinct tricuspid murmur, always more or less undulation in the jugular veins, and lastly a systolic aortic murmur propagated into the carotid arteries. We can seldom trace this gradual development of these murmurs, because when the patient comes under treatment she gets cured, but we can in every case trace their gradual recession in precisely the reverse order to that in which they have just been described to arise. The aortic murmur being the first to vanish, and lastly the venous hum. The aortic murmur was long ago shown by Beau to be due to the large blood-wave sent on by the dilated and hypertrophied heart,¶ and its late development in chlorosis indicates of course

the result of hæmorrhage from the bowels of long standing. All the murmurs of chlorosis were present in this case. He died on May 14th 1880. On *post-mortem* examination the whole surface of the body was blanched and anæmic, rigor slight. The heart weighed  $12\frac{1}{2}$  oz., its muscular fibre on both sides was very flabby and anæmic, not fatty. The aortic valves were competent; the cone diameter of the aortic orifice was 1 inch, of the mitral 1.5, of the pulmonary artery 1.2, and of the tricuspid 2 inches. The left ventricle measured 3 inches in length, its wall from  $\frac{1}{4}$  to  $\frac{1}{2}$  inch in thickness. The right ventricle measured 4 inches in length, its wall  $\frac{1}{2}$  of an inch in thickness. All the other organs were blanched, no special cause of death was found.

\* *Op. cit.* p. 169, *vide* also his *Traité d'Auscultation*, Paris, 1856, pp. 355 and 366.

† *Lehrbuch der Krankheiten des Herzens*, Wien, 1857, ss. 88 and 246.

‡ *Krankheiten des Herzens*, 2. aufl., Erlangen, 1867, s. 172.

§ *Handbuch der Pathologie und Therapie*, Stuttgart, 1856, Bd. iv. s. 354.

|| *Archiv der Heilkunde*, 1863, s. 47; and *Gazette Hebdomadaire*, 1863, p. 262.

¶ *Traité d'Auscultation*, pp. 366 and 565. The history of "The Position and Mechanism of the Hæmic Murmur," in all its forms, will be found detailed in a paper by myself in the *Lancet*, September, 1877, p. 383.



the late development of the conditions upon which it depends, which are much more serious than those found in the earlier stages, though still, as every one acknowledges, within the range of curability. But dilatation with hypertrophy is not always curable in one set of conditions, and always incurable in another, the degree of curability depends partly on the amount of dilatation present, to some extent on its cause, and partly also on the skill of the physician. If the cause be curable, the result may be remedied by nature or more certainly by art. And I know some, who many years ago were believed by competent observers to labour under incurable disease of the heart, who for years have been perfectly free of any trace of such disease; while in my own experience, many have been rescued from such a condition by skilful use of remedies, and I shall presently relate the histories of a few of these. But to this hopeful view there is also a reverse, and it has fallen to my lot to see sufferers from incurable cardiac disease, who, not many years previously, had been assured by most skilful physicians that they had only a functional ailment, out of which they would grow; while I myself have occasionally failed to cure a disease which to my thinking ought to have been remediable. We cannot doubt that the latter class of cases will be much diminished in number, by a due recognition of the fact that the terms functional and organic are convertible, when applied to dilatation of the heart, and signify merely an early and a late stage of the same disease.

I have already mentioned that the basic position of the systolic murmur in the earlier stages of chlorosis is due to the impinging of the fluid veins found at the mitral orifice upon the wall of the dilated auricle, and to the fact that the dilated appendix of the left auricle comes to the surface at the base of the heart, just outside of the pulmonary artery. In this situation, the dilated appendix not infrequently gives rise to so distinct a pulsation that its movements can be traced by the cardiograph, and the history of several such cases has been published, and their cardiograms figured by my



former resident, Dr George Gibson,\* while the pulsation is so well marked and forcible in some of these cases, that the late Dr Hughes Bennett sent me on one occasion a case of chlorosis as a case of aortic aneurism. It is three years since I last saw this woman, and she was then perfectly well.† It is no doubt somewhat puzzling to understand how a murmur of mitral regurgitation comes to be heard at the base, over the auricular appendix, instead of in its ordinary position. This difficulty has not, however, prevented Naunyn's view from being accepted as valid in cases of ordinary mitral regurgitation, by those well able to judge. The application of this view to the explanation of the murmurs of chlorosis, seems to me the only method of escape from the present chaotic state of medical opinion in regard to their origin. It is based upon a condition of heart long known to exist in chlorosis, and it legitimately and logically connects the various murmurs as they arise with that condition and with those consecutive changes which are known to occur in its progress. Furthermore, it seems to me that Naunyn's view is even more applicable in chlorosis than in any other form of heart affection, because the essential cardiac lesion in chlorosis is muscular relaxation and progressive dilatation, hence at a comparatively early stage of the disease the dilated right ventricle has separated the left ventricle from the chest-wall, while the dilated appendix of the left auricle has been *pari passu* brought into closer contact with it. The fluid veins formed in the early stage of chlorotic regurgitation are of low tension and but little force, hence the vibrations they originate are but slightly propagated to the left ventricle, and only with difficulty from it to the chest-wall in the mitral area, where they are heard as an impure first sound. But on the other hand these vibrations are readily communicated to the wall of the auricle on which

\* *Lancet*, September 1877, p. 418; *vide* also paper on "The Rhythm of Auricular Impulses," by the same author, in the *Ed. Med. Jour.* October 1877, p. 299, and May 1878, p. 1012.

† Ellen C., admitted to Ward XIII. Nov. 4th 1869; *vide* also *Lancet*, September 1877, p. 386.

these fluid veins impinge, and are easily transmitted to the chest-wall with which the auricular appendix is in contact, becoming audible in the auricular area as a distinct murmur. By and by, as the regurgitation increases, and the ventricle hypertrophies, these fluid veins gain force sufficient to be communicated through the ventricle also, hence in the later stages of chlorosis we have a mitral murmur associated with the auricular one. It is however quite possible that this murmur in the mitral area is really tricuspid, due to the increased dilatation of the right ventricle, the apex of which may even occupy the mitral area; this not infrequently occurs in mitral stenosis, it is not an improbable event in chlorosis, and it is of little consequence which explanation we accept, the actual truth probably embracing both conclusions, being sometimes due to the one cause and sometimes to the other.

In the person of Helen Christison, Ward XIII. bed 9, admitted 12th February 1875, you have had an excellent opportunity of observing the gradual and consecutive disappearance of all the murmurs described, coincident with the restoration of the patient to perfect health.

The central lesion, so far as the circulation is concerned, upon which all these phenomena depend, is certainly an alteration of the blood revealed by a venous murmur.

Slight venous murmur is often present without other signs of mischief, but when at all developed it is invariably associated with an auricular murmur, and therefore with some degree of cardiac dilatation. Chlorotic murmurs in the mitral area are more rare; usually there is little more than an impurity of the first sound, but occasionally the murmur is exceptionally loud and distinct, and if associated with a previous history of rheumatism, it may be impossible to decide—apart from the results of treatment—whether the murmur is due to chlorosis or to actual valvular disease. The following case affords a striking instance of this:—

CASE XVII.—Elizabeth Heughson, a domestic servant, aged 19, admitted to Ward XIII. on 28th March 1873



examined 29th March. The patient is a middle-sized girl, of fair complexion and anæmic appearance. About two and a half years ago she suffered from rheumatism and pain over the cardiac area; since then she has been liable to occasional rheumatic pains in various parts. She has been much exposed to cold and damp, from the condition of her abode in Oxford, and from having had to take part in the family washing. Her present illness came on in November last as simple weakness, which since then has been gradually increasing. She has not menstruated for the last two periods; menstruation previously had been scanty. Except the circulatory, the other systems are normal, only her digestion is feeble, and her abdomen flatulent. The cardiac apex is felt to beat somewhat diffusely between the fifth and sixth ribs, two inches and three-quarters from the left edge of the sternum. The cardiac dulness, one inch from the left edge of the sternum, commences at the upper edge of the third rib and extends downwards to the liver dulness; at the fourth rib the transverse dulness begins one-quarter of an inch to the right of the sternum, and extends across for a distance of five inches and one-quarter. In the mitral area there is a loud blowing systolic murmur, which is propagated entirely round to beneath the angle of the scapula, and is followed by an accentuated second sound. In the aortic area the first sound is replaced by a murmur, followed by a weakened second sound. In the pulmonary area the first sound is also replaced by a murmur, which is loudest at one inch and a half from the left edge of the sternum, and is followed by a distinctly accentuated second sound. The pulse is 108, small and feeble; a loud bruit de diable is audible in the jugular veins of the right side. She was treated by various preparations of iron, at first with digitalis, latterly in the form of the ammonio-citrate without digitalis. Under this treatment she improved in strength and regained a more healthy appearance, her palpitations almost entirely ceased, but her murmur remained the same. On the 23d of May she was sent to the Convalescent Hospital, whence she returned for examination



on the 19th of June; she was found to have completely regained all the appearance of health, her cardiac murmurs had entirely disappeared, the accentuation of the pulmonary second sound was gone, and the venous hum had also ceased. This case was one in which, though the chlorosis was well marked, yet the history of rheumatism was so distinct that we dared not hope for complete restoration. The result of the treatment showed that the valves had been quite unaffected, and that the whole of the very striking phenomena had been due to simple dilatation of the left ventricle, the result of chlorosis; the absence of any remaining accentuation of the pulmonary second sound conclusively proving that this was a case of actual cure by contraction of the cardiac cavity, and not simply a case of the vanishing of a murmur, the lesion remaining.

I need not say how frequently during the course of acute rheumatism we find a systolic mitral, an auricular or so-called pulmonary, and an aortic murmur developed—the mitral sometimes alone, the auricular occasionally by itself, but never an aortic murmur without one or other of the before-mentioned murmurs coexisting, all these phenomena disappearing on the return of health. After what has been already said, the reason for this is obvious; the defective nutrition of the cardiac muscle, depending on the depraved constitution of the blood, has resulted in relaxation of the muscular tissue and dilatation of the ventricular cavity, this producing imperfect closure of the mitral valve, and hence a mitral murmur which has its position of maximum intensity at one time in the mitral area, and at another in the auricular area, according to circumstances which have been referred to, while these murmurs are propagated in greater or less intensity into the pulmonary and aortic areas. When the normal condition of the blood is restored, the cardiac muscle recovers its tone, the mitral valve again closes perfectly, and all signs of a cardiac affection disappear. Should the fibrous tissue of the valves or of the cordæ tendinæ become thickened or shrivelled as the result of the

rheumatic process, then all the signs of incurable mitral disease become developed ; sometimes uninterruptedly consecutive to those of the curable form ; at others, the curable form of the disease is temporarily recovered from, and the incurable form subsequently developed after an uncertain interval of apparent health, sometimes apparently as the result of the primary rheumatic affection, at others, more evidently caused by the persistence of the rheumatic disease in a chronic, sub-acute, or chronically recurrent form.

Mitral murmurs of a curable character are frequently found associated with chorea ; and though from the close connection which subsists between this disease and rheumatism,\* murmurs originating during its course occasionally become permanent and incurable, yet in a large proportion of these cases the murmurs are curable, and permanently disappear after the patient's restoration to health. In such cases it has been usual to ascribe the occurrence of these mitral murmurs to irregular action of the heart, the result of clonic spasm of some part of the cardiac muscle, particularly one or other of the muscoli papillares, similar in nature to the clonic spasms which in this disease affect the voluntary muscles.† Against this supposition, however, we have the facts, that while the theory of clonic spasm of any part of the cardiac muscle might account for an intermittent murmur, it is not sufficient to explain a mitral murmur, which, while it lasts, is constant and unchanging ; for such has been its character in my own experience ; and I know of no recorded observations which controvert this view. Besides, "there is no good proof that involuntary muscular organs participate in the muscular dis-

\* After all that has been so well written in regard to this matter, particularly by Dr James Begbie in his excellent *Contribution to Practical Medicine*, Edinburgh, 1862, p. 68, &c., it seems superfluous to give additional evidence, yet I may be permitted to add that I have repeatedly seen patients labouring under chorea, in whom the convulsive movements alternated with rheumatic inflammation of the joints.

† Walshe *On Diseases of the Heart and Great Vessels*, 3d ed. London, 1862, p. 96.



order,"\* while "the admitted co-existence of rhythmical action of the substance of the heart with this alleged tetanic and irregular contraction of the papillary muscles, which, as proved by dissection, are directly continuous with the fibres of the ventricular walls, constitutes a still stronger objection to this theory."† Especially if we "bear in mind the unity of the nerve centres, and the community of nerve-distribution enjoyed by both portions of the same fibres."‡ On the other hand, the same deprivation of the blood, and consequent relaxation of the muscular tissue, which is so marked in chlorosis and acute rheumatism, is even more exquisitely developed in chorea; hence, there is no difficulty in referring the continuous mitral murmur in chorea to the same cause which has been already shown to be capable of producing it in those diseases referred to.

There is hardly any form of febrile disease in which so-called hæmic cardiac murmurs have not occasionally been heard. In scarlatina, relapsing and enteric fevers, they are of common occurrence; in erysipelas, smallpox, measles, and probably in all other febrile diseases, they are occasionally heard; while in true typhus such murmurs are somewhat rare.§ When we consider that relaxation and softening of all the muscular tissues, both voluntary and involuntary, is common in all protracted forms of febrile disease, and when we also reflect that this alteration of the muscular tissue depends mainly upon altered nutrition, and must therefore vary considerably according to the previous condition of the patient in

\* Kirke's *Medical Times and Gazette*, 1863, p. 637.

† Hayden, *op. cit.* p. 263.

‡ *Loc. cit.*

§ For a history of the state of the heart in typhus and other fevers, and for an account of the murmurs which occur during their course, I beg to refer to Dr Stoke's admirable chapter "On the Condition of the Heart in Typhus Fever," in his work on *Diseases of the Heart and Aorta*, Dublin, 1854, p. 366; and for an account of the morbid changes which are found in both the voluntary and involuntary muscles of fever patients, I may refer to Zenker's Memoir, published at Leipzig in 1864, or to Murchison, on *The Continued Fevers of Great Britain*, London, 1871, pp. 248-9.



this respect, we see sufficient reason why in ill-nourished and anæmic patients a less protracted disease should give rise to phenomena which in better nourished patients are only occasionally observed, even when they have been exposed to a more severe and protracted disease. Hence the want of constancy in the cardiac phenomena referred to in every disease; hence, too, their probably greater frequency in hospital patients. The alleged infrequency of such a murmur in typhus patients is readily understood, when we consider that to produce sonorous fluid veins a certain energy of the propelling force is requisite, and remember, also, that the cardiac muscle is frequently so enfeebled in this disease that murmurs due to actual disease of the valves disappear during its course, and reappear as the patient convalesces and the heart reacquires sufficient force.\* Probably enough, careful auscultation of the auricular area in every case of typhus would reveal a systolic murmur there as the first and earliest indication of failing cardiac power; if this be not the case, the absence of this murmur can only be referred to the prevalence of anæmia over mere spanæmia in most cases of this disease—a condition exactly the reverse of that found in chlorosis.

In all febrile diseases, whatever their character, the sequence of the phenomena, as observed by myself, has been precisely similar to that observed in chlorosis. And you will readily perceive how important a matter this is; for if these chlorotic and febrile murmurs be actually due to dilatation of the heart, and if it be also true, as it undoubtedly is, that all, or, at all events, all but an infinitesimal proportion of such cases make a perfect recovery,† then we must very considerably

\* Stokes, *op. cit.* p. 446; recapitulation, 17, &c.

† We all know that chlorotic murmurs ordinarily result in perfect cure, and in regard to those occurring in the course of fevers, Stokes says, "Of many hundreds of cases of weak and softened hearts observed during the last twelve years, we cannot adduce a single instance of organic disease of the heart which could be traced to any injury done pending the typhous affection."—*Op cit.* page 374, and we must remember that under the term "typhous" Stokes includes not typhus only, but also enteric and relapsing fevers.

enlarge the area of curable cardiac affections, and there can be no doubt that this is the case. I do not now of course refer to the simple vanishing of murmurs, due to organic valvular lesions, which are permanent, as can be readily proved; in such cases, of course, there is and can be no real cure, but to those numerous cases of positive dilatation of the ventricular cavities, which may range, as we have seen, from the most unimportant case of chlorosis up to a very serious amount of cardiac dilatation, the curability of these being simply a question of degree. In chlorotic and febrile dilatation, cure, as we know, is the rule; in dilatation from other causes, it is perhaps the exception, but it may always be aimed at, and is not infrequently attained. In simple dilatation of the right ventricle, the result of bronchitis, the cure of the bronchitis is usually followed by cure of the cardiac affection, but to this we shall recur in speaking of diseases of the right side of the heart. In dilatation of the left ventricle from ordinary causes, usually occurring at or beyond the middle period of life, and depending upon arterial atherosclerosis, so often euphemistically termed the gouty diathesis, or upon strenuous exertion accompanied by the free use of alcohol, the patient seldom comes under treatment till the right ventricle is also affected secondarily. Yet, even in these cases, very great improvement is of common occurrence, and in a few a positive cure—or at all events one of some permanence—is actually attained. I may give the following cases as very interesting and instructive examples of this; the cases are of course very much condensed, only the salient points in the cardiac lesion being referred to:—

CASE XVIII.—Henry Welsh, a labourer, aged 61, admitted to Ward V., 17th November 1874, complaining of pain in the chest, shortness of breath, swelling of the stomach, and occasional palpitation. About thirty years ago this patient had an attack of rheumatic fever which he says lasted twenty weeks. For the last ten years he has had occasional pains in the left haunch and arm, which he calls rheumatic; with



these exceptions, his health was perfect up to last August, when he was suddenly seized with severe pain in the front of the chest, accompanied by shortness of breath and expectoration; this lasted about a week. After this recovery, on attempting to work, he found that the slightest exertion produced breathlessness and much feeling of weakness; the pain in the chest felt at first abated, but left behind it a soreness which still continues. About a month after his illness, he first noticed swelling of his feet at night, and since then he has remained much in the same condition. The patient is a well-built powerful-looking man, nearly six feet in height; his muscles are, however, soft, and he says he has lost much flesh. Now he can lie in any posture, but till six weeks ago he required to have his head raised on account of breathlessness. Expression of face natural, skin natural, temperature  $98.6^{\circ}$ . The soreness complained of is referred by patient to a point about the middle of the sternum; it is always present, and though not very severe, it becomes worse upon any exertion. He has no dyspnoea when at rest, but on making any exertion a choking sensation is perceived, referred by him to the part where pain is felt. At first, was much troubled with palpitation, but this is not now so frequent. Pulse 92, composed of alternate strong and weak beats; at times the force of each beat is tolerably uniform, but accompanied by intermissions and occasional double beats. The veins generally are not distended, the external jugulars are slightly so, and pulsate distinctly, but do not fill from below on pressure being made on them with the finger. The cardiac apex beats between the fifth and sixth ribs, four and a half inches from the median line; no other pulsation is visible. On palpation, the cardiac impulse is felt to be feeble and irregular, both as to time and force. On the left side, one inch from the sternum, the cardiac dulness does not rise above the third rib. Transversely, in the plane of the fourth costal cartilage, the dulness extends from half an inch to the right of the sternum to four inches to the left of the left edge of that bone. On



auscultation in the mitral area a high-pitched systolic murmur is heard, accompanied in three beats out of four by a partially closed first sound; in the fourth the murmur alone is audible. The maximum intensity of this murmur is in the mitral area, but it is propagated distinctly round to the angle of the left scapula. In the mitral area this murmur is followed by a somewhat obscure second sound. In the tricuspid area a loud but low-pitched systolic murmur is distinctly audible, accompanied by some portion of a first sound, and followed by a second sound, which is sharper than that in the mitral area. At the base the first sound is closed, the pulmonary sound markedly accentuated. There is no venous murmur. This patient gradually improved under treatment, till, on his discharge on 18th February 1875, the only evidence of cardiac disease present was a slight impurity of the first sound in the mitral area, and an almost imperceptible accentuation of the pulmonary second. The tricuspid murmur had entirely disappeared.

Time alone can prove whether in this case there was slight damage to the mitral valve, dating from his rheumatic attack thirty years ago, or whether, as seems probable, his sole lesion was dilatation of both ventricles, dating from his febrile—probably bronchitic—attack of August last. In any case, his improvement was both marked and remarkable, and the contraction of both ventricles, but particularly of the right one unmistakable.

In the following case, the nature of the lesion is less open to question, and the improvement even more extraordinary:—

CASE XIX.—Euphemia Lawson, a domestic servant, admitted into Ward XIII. on the 26th September 1872, labouring under breathlessness and swelling of the lower extremities. In this case there was forcible pulsation at the lower part of the sternum, pulsation of the jugular veins, and slight increase of the transverse dulness of the heart. In the mitral area a blowing murmur, and in the tricuspid area a similar murmur of a somewhat lower pitch completely replaced the first

sound. These murmurs were propagated into the aortic and pulmonary areas; the aortic second sound was weakened; the pulmonary second much accentuated. She never had any rheumatism whatever, but had hard work, and was rather given to the abuse of stimulants. About three months after admission, at the hour of visit, we were suddenly called to her bedside to find her completely unconscious, and paralysed on the left side. The unconsciousness passed off within half an hour. The paralysis of the arm ceased to be noticeable in a few days; and, after the lapse of some weeks, the paralysis of the leg was only revealed by a slight dragging, which ultimately quite disappeared. Under treatment, all her cardiac symptoms also vanished; and, at her discharge, on the 26th of March 1873, the only detectable sign of what had originally appeared to be a most serious disease, was a slight blunting of the first sound; all the murmurs had quite disappeared, and even the accentuation of the pulmonary second could no longer be detected. She was so well that she left the Infirmary to go direct to service, and in service she has been ever since. I have during the time that has elapsed had occasion to see this patient frequently, and have always found her heart as it was when she was discharged.\*

\* August 19th 1875.—Euphemia Lawson has again come under treatment. She has œdema of the lower extremities to no great extent, breathlessness, and some rhonchi and frothy watery sputa, the latter coming on after admission. Her heart is by a repetition of the original causes again dilated, she has again pulsation beneath the lower part of the sternum, no perceptible apex beat, and all the other symptoms as above described; the pulsation of the heart is however more forcible than it was, obviously hypertrophy is beginning to become associated with the dilatation, this is to say, a heart rehabilitated by hypertrophic contraction has again become dilated, the hypertrophy continuing; now therefore we have the commencement of what is termed the dilated gouty heart. We shall still I believe be able to rehabilitate it, but less effectually than formerly, and slighter disturbance will bring back the dilatation, until at last we have the usual termination of such cases, general dropsy and death from asthenia. It is interesting to note that she has now not the slightest trace of any paralysis remaining.—Nov. 4th 1875.—It is perhaps even more interesting to have to record that this patient improved so much under treatment, that she was discharged on September 19th with her heart once more completely rehabilitated. Euphemia Lawson came again under treatment on February



But perhaps the most remarkable case of cure of a dilated heart was that of Mrs G.,\* who was admitted to Ward XIII. in the beginning of 1877, labouring under intense spanæmia, due to an abortion occurring in the course of typhoid fever; she had a loud venous hum, and a distinct pulsation accompanied by a loud systolic murmur in the auricular area, one inch and a half to the left of the sternum in the second intercostal space. After some months of treatment she was dismissed cured, all her symptoms and physical signs having disappeared. Her home was not all it ought to have been, and in a few months she returned with a relapse of all her symptoms. She was advised to come into hospital, but preferring to remain at home, she was dismissed with a prescription for a ferruginous tonic. She did not improve, however, and was readmitted to Ward XV. in October 1877, labouring under general dropsy, and passing only a small quantity of urine deeply tinged with blood. Her heart, particularly the right side, was greatly dilated, there was a loud systolic murmur in all the cardiac areas, and well-marked jugular pulsation. The essential part of her treatment consisted in the administration of digitalis, in large doses for short periods, twenty to thirty minims of the tincture or a proportionate dose of the infusion being given every four hours till a decided effect was produced on the heart. The digitalis was then pretermitted for a time, and during this interval iron, iron and quinine, or iron and arsenic were administered to improve the state of her blood. In this way her strength was gradually improved, her dropsy removed,

9th 1876, and after much suffering from ever recurring embolisms, she died on April 29th 1876. Her heart was much dilated and somewhat hypertrophied, in it were numerous thrombi. The valves, mitral and aortic, were slightly atherosed, otherwise natural. During her last period of residence in hospital she presented a rare form of cardiac irregularity of the bigeminal type, the right and left ventricle alternately predominating, and the carotid artery and jugular vein pulsating alternately. This case has been published *in extenso* along with another similar one by my then resident Dr C. S. Roy, in the *Ed. Med. Jour.* Jan. 1878, p. 594.

\* This case as it was when first admitted will be found narrated in full in the *Ed. Med. Jour.* October 1877, p. 302, by my resident Dr George Gibson, who has also given a cardiogram of her auricular pulsation.



and her heart contracted, so that in June 1878 she was exhibited to the Medico Chirurgical Society without a trace of murmur, without any accentuation of the pulmonary second, and with only a slight hypertrophy of the heart remaining as the sole indication of serious disease which had existed.\* I saw this patient lately (June 1880); she is in perfect health, and presents no indication of heart trouble beyond a blunt first sound, and a trifling increase in the force of the apex beat.

You will see, then, that cardiac dilatation is by no means a hopeless disease; that it is invariably accompanied by mitral regurgitation, which, in more severe forms, is associated with tricuspid regurgitation also; that in its slighter forms, it is readily curable, and is almost invariably cured; and that, even in its more severe forms, it may occasionally be cured and is always susceptible of considerable amelioration, the only exceptions being those neglected cases associated with notable eccentric hypertrophy, forming what is termed the gouty heart.

In simple chlorotic cases, it is sufficient to improve the quality of the blood to secure a perfect cure. For this, as every one knows, there is no remedy equal to iron; and though small doses of ferruginous tonics, especially if continued for a long time, are frequently sufficient, still, the larger the dose given the more rapidly the cure is obtained; and I perfectly agree with Niemeyer, that there is no combination in which iron may be more freely administered than as Bland's pills—two grains and a half of sulphate of iron, and the same quantity of carbonate of potass, made into one pill with mucilage. Three such pills are to be taken three times a day, and the dose is to be increased by one pill each day till five pills are taken three times a day. The remedy to be continued for six weeks from its commencement, when the cure will be complete. This remedy, notwithstanding the large amount of iron given, does not usually constipate the bowels.

\* Vide *Ed. Med. Jour.* June 1878, p. 1120.

When the dilatation is more marked, the tonic action of digitalis on the heart will be also required, and the dose of this drug must vary with the requirements of each case. In serious dilatation it is often necessary to give large doses at regular intervals till a decided effect on the heart is produced, then premitting the digitalis for a time, and giving iron pretty freely during the interval. It is better to avoid giving iron with digitalis, as sickness is then very apt to be produced. In less serious cases small tonic doses of digitalis or digitaline will suffice, and these may be given night and morning, while iron with or without arsenic may be given simultaneously, along with the food. To show how long small doses of digitalis may be continued without risk, and how beneficial is their action, even in apparently untoward circumstances, I may mention that I recently saw an old gentleman, of over 80 years of age, who has been taking these doses of digitalis for more than four years regularly. When first seen, his heart was so weak that its action was almost imperceptible; he had a feeble pulse, and was subject to frequent fainting fits. Now he rarely has an attack of this nature, has a good pulse, and a firm, forcible apex beat.\* Of course, good nourishing food must be given, fresh country air must be recommended, and perfect rest, or at least the avoidance of all but the most moderate exertion. Stimulants in younger patients are best avoided; in elderly patients they are often useful, and occasionally necessary; but must be used with caution, as simple tonics, and never as goads to exertion, otherwise they do an incalculable amount of harm.

\* June 1880. This old gentleman is still alive, and takes his digitalis, but not so regularly as formerly. He was 77 when he first came under treatment, and is now 87.

## LECTURE VII.

## ON TRICUSPID REGURGITATION, CURABLE AND INCURABLE.

GENTLEMEN,—In speaking of the murmurs connected with mitral stenosis, I have already referred to one case of tricuspid stenosis, the existence of which was determined by the results of a *post-mortem* examination.\* Since that time we have had in Ward V. another case in which the mode of propagation of the existing presystolic murmur left no doubt upon my mind that we had to do with stenosis of the tricuspid valve as well as with that of the mitral valve.† Happily we were enabled to discharge this patient somewhat improved; and, so far as I know, he still survives. Two other cases of tricuspid stenosis have also been recorded by Dr Haldane,‡ in one of which the diagnosis was made during life. In little more than ten years, therefore, we have had in the Edinburgh Infirmary four cases of tricuspid stenosis, so that this form of lesion, though infrequent, cannot be ignored. Of course, as I have already mentioned, in regard to the mitral valve,§ stenosis in its most frequent form involves regurgitation also, and, indeed, in two of

\* *Vide* Case IX. p. 121.

† Case of Martin Kelly, aged 16, admitted to bed 6, Ward V. on January 26, 1874; discharged March 19.

‡ *Edinburgh Medical Journal*, September 1864, p. 271. So far as I know these are all that have been recorded; there is every reason to suppose, however, that others have occurred, especially as I am conscious having occasionally seen in the *post-mortem* theatre hearts in which thickening and slight shortening of the segments of the tricuspid valve had occurred.

§ Lecture V. p. 135.



these cases the fact of tricuspid regurgitation was distinctly established during life.\* Of course this form of tricuspid regurgitation is absolutely incurable. I have also already mentioned that tricuspid regurgitation—the result of dilatation—is a frequent concomitant of mitral stenosis, being earlier developed and more remarkable when that has occurred early in life, and is associated with any considerable degree of aortic hypoplasia, forming the almost invariable prelude to the end in all such cases.† In cases such as these the tricuspid regurgitation is always considerable, and its effects upon the systemic venous circulation well marked; it is, of course, incurable, though, as already noted, it may be—especially at first—somewhat modified by treatment, and the inevitable end postponed; the degree in which this can be done depending upon the amount of stenosis present, as well as upon that of the aortic hypoplasia. But, as a rule, the dilated condition of the right side is persistent, and the hypertrophy ere long becomes considerable, so much so that at times the right ventricle forms the apex of the heart, and the left ventricle appears like a mere appendix attached to it.‡

The physical signs upon which we rely as certain indications of tricuspid regurgitation are few, simple, and easily recognised. First, as the result of the dilatation of the right ventricle, and of its anatomical position in front of the left ventricle, we have a disappearance of the apex beat from its usual position, and its replacement by an impulse just beneath the lower end of the sternum, which is more or less heaving and forcible in proportion to the degree of hypertrophy present. Even in perfect health, if we place one finger on the apex beat, the palm of the hand resting on the lower part of the sternum, and hold our breath as long as possible, the apex beat will be felt

\* *Edinburgh Medical Journal*, *loc cit.*, pp. 271, 272.

† Lecture V. p. 157.

‡ Case XIV. p. 142. Many similar cases have also occurred, and indeed I may as well now say that all the cases recorded are selected representative cases, each one must therefore be taken as a sample of a group, and not merely as a single instance of any peculiarity.

gradually to become fainter, and may quite disappear, at the same time that an obscure pulsation is felt just beneath the sternum. The reason for this is that as the lungs become congested, the blood, unable to pass onwards, accumulates in the right ventricle, gradually dilating it, swelling it out like a water cushion in front of the left ventricle, which is thus pushed deeper into the cavity of the chest, consequently the apex fails to reach the chest-wall, and ceases to be felt.

On percussion we have an increased breadth of dulness at the level of the fourth rib, the result, however, rather of secondary dilatation of the right auricle than of any increased breadth of the ventricle.

On auscultation in the tricuspid area, the sternal end of the fourth, fifth, and sixth ribs, we usually have a loud systolic murmur, which is propagated with more or less distinctness into all the other cardiac areas, but has its position of maximum intensity at the spot referred to. When mitral regurgitation coexists with tricuspid regurgitation, we have two foci of maximum intensity for the systolic murmur, one in the tricuspid area, and one in the mitral area; there is often also a difference in pitch between the two murmurs, but sometimes the one and sometimes the other has the higher pitch. Like most of the cardiac murmurs, the systolic tricuspid murmur is occasionally absent, even when all the other signs of tricuspid regurgitation are certainly present, the murmur being always the least reliable sign.

Even without any notable regurgitation through the tricuspid valve, if pulmonary congestion, and consequent overfilling of the right ventricle, has persisted for some time, we have as its necessary result great congestion of the right auricle and of the systemic veins. This naturally attracts attention in the right external jugular vein, which in such circumstances is always more or less turgid, and frequently undulates with a wavy motion communicated to it by the systolic percussion of the turgid tricuspid valve through the medium of the blood in the over distended auricle, which has all the elasticity of



a compressed fluid. When, however, from long continued tricuspid regurgitation, the distention of the veins has become so great as to render nugatory the action of the valve at the root of the external jugular, where it enters the subclavian, then we have distinct systolic pulsation in the external jugular vein, the vein filling from below synchronous with each ventricular systole. This pulsation is more or less evident at all times, according to the size and visibility of the vein, but is always best marked in elderly persons, in whom the external veins are usually larger, and the quantity of adipose tissue covering them less than in younger individuals. Compression of the vein at the middle of the neck, by stopping the downward flow of blood, makes this systolic reflux more visible and remarkable. Jugular pulsation is usually present when we have a systolic tricuspid murmur, and it is sometimes visible when no murmur is audible; still, as it is always the result of great previous venous turgescence, it may be always accepted—even in the absence of murmur—as an indication of existing tricuspid regurgitation, provided the other signs already referred to be also present. Should, however, the substernal pulsation be absent or slight, it may be inferred as possible that the jugular pulsation may only be the remanent result of a pre-existing tricuspid regurgitation, after the valve has been perfectly rehabilitated, what would otherwise have been only an undulation, retaining now its distinctly pulsatory movement. It is thus obvious that jugular pulsation is always a sign of existing or of pre-existing and readily reproducible tricuspid regurgitation, and is, therefore, a sign of considerable importance. But the jugular vein is not the only one which is filled by the systolic reflux, and from the absence of valves, the considerable size and the depending position of the vena cava inferior, the regurgitant blood must flow with great freedom and directness down that vessel into the liver. Hence hepatic pulsation is a phenomenon which may be observed, in many cases at least, even prior to the occurrence of jugular pulsation. Hepatic pulsation may,



however, be caused in a threefold manner. Firstly, we may have simply a systolic depression of the liver, chiefly the left lobe, synchronous with the contraction of the ventricles, and apt to be regarded as the pulsation of the heart itself. Secondly, we may have a heaving of the whole mass of the liver, due to a movement communicated to it by an aneurism of the descending aorta, or by the vena cava, which is often enormously dilated.\* This pulsation is not synchronous with the ventricular systole. And thirdly, we may have a distinctly expansile pulsatory movement also communicated to the whole mass of the liver, by the systolic pulsation of the hepatic veins ramifying within it; this pulsation is also delayed, and is not synchronous with the ventricular systole. The two latter modes in which hepatic pulsation may be produced, are signs of a much more advanced and more serious degree of cardiac dilatation than suffices to produce it in the first-mentioned manner; hepatic pulsation which is retarded and is not synchronous with the cardiac systole is, therefore, an important diagnostic phenomenon. There is no doubt that these forms of hepatic pulsation have not usually been sufficiently discriminated, and that hepatic pulsation has been too generally referred simply to the ventricular impulse, even when the absence of synchronism has been distinctly recognized. Thus Allan Burns refers to a most furious pulsation in the epigastric region, "produced," as he says, "entirely by the dilated heart," in which, from the absence of synchronism between the heart and the pulsating tumour, no one ever doubted but that the patient had an aneurism of the coeliac artery.† In very rare cases pulsation of the superficial veins has been observed as the result of tricuspid regurgitation, but it is so extremely uncommon as to be unimportant.

It is usually said that tricuspid regurgitation alters or removes the preceding accentuation of the pulmonary second sound. It certainly must alter it, but inasmuch as we have not before us the two conditions simultaneously, we never can

\* *Vide* p. 8.

† *Op. cit.*, p. 264.

say to what extent it is changed. But it never does and never can altogether remove it, because the essential cause of the production and maintenance of tricuspid regurgitation is increase of the blood pressure within the pulmonary circuit; so long, therefore, as tricuspid regurgitation persists, the pulmonary second sound will be always accentuated relative to its normal and to the aortic second, which from the same cause must be enfeebled.

As already said, mitral stenosis is a frequent cause of serious tricuspid regurgitation, and the earlier in life the stenosis occurs the more rapidly, as a rule, the tricuspid regurgitation follows, and the more serious the prognosis. But any intra-cardiac obstruction in the left heart to the circulation may give rise to tricuspid regurgitation, hence simple mitral regurgitation from dilatation of the left ventricle is not infrequently accompanied by this form of disease. This, however, rarely occurs early in life, is never relatively so excessive, and the dilatation and hypertrophy of the right side usually progress *pari passu* with the similar affection of the left side. At first by treatment we may be able to rehabilitate the tricuspid valve; but unless we can also rehabilitate the mitral, we only gain a temporary postponement of the end. Still this end is never so rapidly reached as in great mitral stenosis; and from the simultaneous implication of both ventricles, and the lengthened duration of the disease in such cases, the whole heart frequently becomes enlarged to a veritable *cor bovinum*,\* before the gradual transference of the blood from the arteries to the veins culminates first in general dropsy and lastly in fatal asthenia, and careful treatment may do much to mitigate the inevitable evils and to postpone the end. Great obstruction at the aortic orifice has, of course, a similar effect with mitral stenosis, but such extreme aortic constriction is really so rare as to be of little practical importance as a cause of

\* An adjective, by the way, which, though unusual, is yet strictly classical, as we find "*Medulla bovina*" spoken of, Theodor. Priscian, *De dicta*, c. 15.



tricuspid regurgitation. On the other hand, aortic regurgitation is frequent enough, but its own peculiar sources of mortality are such as to anticipate any important alteration of the right ventricle.\* It is only comparatively rarely, therefore, that aortic regurgitation gives rise to tricuspid regurgitation of a serious character.

Next to diseases of the left side of the heart, diseases of the lungs are those which affect the right ventricle, and of those only two are of much importance. Chronic and especially tubercular diseases of the lungs only exceptionally produce any alteration of the right ventricle, because of the simultaneous diminution of the amount of blood circulating. Acute diseases, such as pneumonia, never last long enough to produce any serious alteration of the right heart, and even pleurisy, with great effusion, is seldom sufficiently protracted without so much diminution of the mass of blood as to cause consecutive dilatation of the right side to be a comparatively rare complication. It is otherwise with bronchitis—acute or chronic—in which the obstruction to the circulation is great, lasts sufficiently long, and is accompanied by no diminution of the mass of blood sufficient to nullify its dilating effect upon the right ventricle. In bronchitis, therefore, we have, next to mitral stenosis, the most common cause of considerable tricuspid regurgitation. But inasmuch as bronchitis is a curable disease, the tricuspid regurgitation depending upon it is evanescent, of short duration, and does not always recur even with a return of its cause; because, of course, cardiac dilatation presupposes loss of tone of the cardiac muscle, which may be pyrexial in character, or of defective nutrition either of general or local origin. In the first instance the rehabilitation of the ventricle is generally complete and more or less permanent, while in the second instance any possible rehabilitation largely depends upon the cause of the abnormal nutrition.

Emphysema of the lungs, which is the only other pulmonary

\* *Vide Lecture III., p. 92.*



cause of tricuspid regurgitation,\* is a more serious one, because it necessarily presupposes a greater or less limitation of the area of the pulmonary capillaries, and consequently an obstruction to the circulation as permanent and incurable as that of any equivalent amount of mitral stenosis. Tricuspid regurgitation following emphysema of the lungs is therefore a disease the prognosis of which is of quite as serious an import in regard to curability as that following mitral stenosis; but, as we shall by and by see, the prognosis as to longevity is not by any means so grave. It has, however, certain physical signs peculiar to itself, to which I shall presently refer.

The following case presents a well-marked example of curable tricuspid regurgitation:—

CASE XVII.—William Savage, aged 38, a labourer, admitted to bed 3, Ward V., on March 24, 1870, complaining of great difficulty of breathing, cough, and expectoration. On admission the patient stated that he worked in a quarry, was constantly liable to chills,—the results of exposure to cold winds while heated by hard labour,—that the present attack was the result of one of these chills, and that he had repeatedly suffered from similar attacks, in two of which he had been treated by copious venesections. He was tall and strongly made; his countenance swollen and dusky; he suffered from orthopnoea; his skin was cool; his finger-tips purple. On inspection the apex beat was seen to be absent, and instead of it we had a pulsation beneath the lower part of the sternum and in the scrobiculus cordis. On palpation the chest was felt to expand equally in all directions, and a systolic heaving impulse was felt beneath the lower part of the sternum, the apex beat

\* It is somewhat strange that C. Gerhardt, who distinctly recognises the influence of pulmonary emphysema in producing dilatation and hypertrophy of the right side of the heart, should yet refer the systolic murmur accompanying this to mitral regurgitation, which he supposes to be produced in some extraordinary manner by the defective blood supply in the left side of the heart and the arteries. He speaks of this so-called mitral systolic murmur as "ein zeichen localer anæmie" (*Lehrbuch der Auscultation und Percussion*, Tübingen, 1871, p. 264).

being absent from its usual position. The percussion sound over the chest was normal, except a slight increase of the transverse dulness of the heart, which at the level of the fourth rib commenced nearly one inch to the right of the sternum. On auscultation the chest was found to be so full of moist crepitation and a few rhonchi as completely to obscure the heart's sounds, except that the pulmonary second was heard to be distinctly accentuated. Large jacket poultices of linseed meal were ordered to be applied over the whole chest, and the following prescription :—

<b>R</b>	Ammoniae carbonatis,	.	.	.	3 ii.
	Spiritus chloroformi,	.	.	.	3 iv.
	Decocti senegae,	.	.	.	3 vi.

Sig.—Cochleare magnum quâque quartâ horâ.

March 25.—Slept badly on account of his orthopnoea; whole surface livid, otherwise unchanged. To have infusion of digitalis instead of the senega. March 26.—To-day he was somewhat better, and a systolic murmur could now be heard in the mitral area replacing the first sound. A systolic murmur was also distinctly heard in the tricuspid area, and in the aortic area a murmur replaced the first sound, the closure of the semilunar valves being distinctly heard. Next day, the 27th of March, the lungs were so far free of crepitation that it was possible accurately to differentiate the mitral from the tricuspid murmur, and the patient could now sleep well in the recumbent posture. On the 29th of March the patient felt almost well, had but little cough, his breathing was easier, and colour natural. On 1st April it was noted that though the tricuspid murmur was still distinct, yet the pulsation of the right ventricle was chiefly confined to the lower part of the sternum, and was hardly perceptible in the epigastric region. On 2d April it was noted that the chest was free from crepitation, and that no murmur was audible in any of the cardiac areas. On 9th April he had an attack of pleurisy on the right side, with friction, which was unaccompanied by any return of



his cardiac symptoms. It was, under appropriate treatment,—chiefly poulticing, and the administration of iodide of potassium—recovered from in two or three days; and the patient was discharged well on 18th April. On 15th December of the same year (1870) this patient returned to hospital, complaining of some dyspnoea, cough, pain over the lower part of sternum, and slight pulsation in the epigastrium. On auscultation his cardiac sounds were found to be distinct and free from murmur, the pulmonary second accentuated. Fine crepitation audible over the right base anteriorly. Percussion normal. A jacket poultice and rest were prescribed, and on the 26th of December he was discharged quite well.

Now, the first remark I have to make in regard to this case is that the coexistence of a mitral murmur with a tricuspid one, due to pulmonary obstruction, must certainly be a rare phenomenon, yet it is a possible one; and it is recorded as my distinct impression at the time. If it be correct it is a further and important proof of the influence of febrile muscular relaxation in promoting curable mitral regurgitation, even under circumstances otherwise unfavourable for this development;\* while even if it be a mistake it in no respect invalidates the importance of the case as a remarkable instance of curable tricuspid regurgitation—of which all the other phenomena were present—depending upon pulmonary obstruction of a temporary character. I have selected this case as an example of this affection, mainly because we had in this patient a subsequent opportunity of observing the permanency of the cure. The following case is an equally instructive instance of tricuspid regurgitation, incurable, not because it depended upon dilatation of the right ventricle, but because that dilatation was produced by a permanent and irremediable form of pulmonary obstruction.

CASE XVIII.†—Andrew Laidlaw, a cabinetmaker, aged 49, was admitted into bed 11, Ward V., on 23d October 1874,

\* *Vide* Lecture VI.

† Reported by Mr C. S. Roy, clinical clerk.



complaining of indigestion and breathlessness. He stated that about two years ago he first suffered from shortness of breath while at work, accompanied by a feeling of oppression in his chest, a troublesome cough, spit, and severe pain darting through his chest from before backwards, on the occurrence of any severe paroxysm of coughing. For the relief of this pain mustard poultices were applied with success. He states that he has suffered from rheumatic fever, for which he gives no date, and that, at the age of 19, he had an attack of measles, since which he has never been free from cough and spit. His personal and family history are unimportant; he has always had a comfortable home, and from the nature of his occupation he has not been much exposed to the weather. The patient is a man of fair muscular development, 5 feet 7 inches in height; the expression of his face is natural. He suffers from a certain amount of dyspnoea whenever he makes any exertion; he has a good deal of cough, which occasions him a certain amount of pain, and is accompanied by the daily expectoration of about five ounces of muco-purulent sputa. Two years ago he had also slight hæmoptysis. His respirations amount to 20 per minute. His pulse is 80, soft and full, and he occasionally suffers from palpitation and pain at the præcordia. On inspection his chest-walls were seen to be emaciated, and the intercostal spaces somewhat distended, giving to the thorax a rounded barrel-shaped appearance. The rhythmical movements of the thoracic walls appeared somewhat restricted, and were unbroken by any cardiac tapping in the normal position; but a distinct localised pulsation was visible in the epigastric region, from which a pulsatory tremor passed over the liver. On palpation both sides of the chest were found to expand equally but imperfectly, especially over the upper part of the chest, and on passing a tape round the thorax at the level of the fourth rib, the difference between deep expiration and full inspiration was found to be only half an inch. No cardiac impulse was to be felt in any part of the præcordia, but a distinct limited pulsation was

felt in the left side of the epigastric region, synchronous with which there was felt a distinct but somewhat tremulous pulsation extending over what was subsequently ascertained to be the area occupied by the liver. On percussion anteriorly hyper-resonance was found over both lungs, particularly their upper parts; on the right side in the nipple line this hyper-resonance reached down to the upper border of the fifth rib, and beneath that the liver dulness extended for five inches; on the left side, one inch from the edge of the sternum, the percussion note was hyper-resonant down to the third interspace, beneath which, at the level of the fourth rib, it gradually rose in pitch and lessened in duration down to the level of the sixth rib; below this the tympanitic note of the stomach alone was heard. On percussing across the sternum, at the level of the fourth rib, the cardiac dulness was observed to commence at the right edge of the sternum, and extended across to the left nipple, a distance of four inches. Posteriorly, the percussion note was not only lower in pitch and of longer duration than usual (hyper-resonant), but could also be carried down to beneath the twelfth dorsal vertebra, though on the right side the percussion note began to shorten in duration and to heighten in pitch directly we passed the upper border of the sixth rib. On auscultation over the lungs the respiration was everywhere found to be indeterminate, presenting the character of an imperfect inspiratory vesicular murmur followed by prolonged expiration, accompanied by sibilant rhonchi. On listening in the normal cardiac areas, the heart's sounds were somewhat faintly audible, and no murmurs were to be heard. At the base the aortic and pulmonary second sounds were more distinctly audible at the sternal ends of the third rib on the right side, and of the fourth rib on the left side, than in their normal position at the sternal end of the second right and third left ribs. Over the pulsating tumour in the epigastric region a soft blowing murmur was heard replacing the first sound. His tongue was large, pale, flabby, and indented by the teeth, which were much decayed; his



appetite was indifferent, deglutition perfect, and he occasionally vomited what food he took. On palpation and by percussion the liver was felt to come down beneath the ribs, and it also lay beneath the pulsating epigastric tumour. The splenic dulness was not enlarged. The urine was of a dark sherry colour, specific gravity 1022, acid reaction, deposited a slight mucous cloud on standing, and contained nothing abnormal. He occasionally suffered from severe frontal headache, had sometimes been deaf, and since, shortly after the commencement of his last illness, he had been obliged to use spectacles, but had no other symptoms of nervous change. After about a week's treatment, consisting of five grains of carbonate of ammonia in half an ounce of infusion of digitalis every four hours, an impure first sound was occasionally—about every third beat—to be heard over the epigastric pulsation; but the heart never became any more perfectly rehabilitated, and, though the bronchitis speedily disappeared, no treatment had any further effect on the condition of the heart. On February 4, 1875, he was discharged, free from bronchitic complication, but otherwise in *statu quo*.

This very interesting case was sent to the Infirmary, supposed to be labouring under an aneurism of the abdominal aorta; but an aneurism of the aorta reaching the surface in the position of the pulsating tumour here, just below the ribs in the epigastric region, is usually more distinctly distensible in the character of its pulsation, and this, as well as the accompanying murmur, are always delayed beyond the systolic pulsation and sound of the heart, which in this case they were not. But one conclusion, therefore, could be arrived at, and that was that the right ventricle was in this patient depressed below its usual position, that it was dilated, and the tricuspid valve incompetent. And the physical signs already detailed not only agreed in confirming this, but they also informed us that the cause of this depression was of such a kind as to entail tricuspid regurgitation of a permanently incurable character.



#### DISEASES OF THE HEART.

The alterations of the percussion note in the precordium would not of itself be sufficient to determine depression of the heart, nor would the coexistence of a pulsating tumour in the epigastrium at all aid in establishing this diagnosis; because the first might be solely due to emphysematous hyper-resonance of the lung obscuring the cardiac dulness at its upper part, while the latter, even if it were distinctly ascertained to be cardiac and not simply hepatic pulsation, might solely depend upon great dilatation of the right ventricle. But when we find that the vertical cardiac dulness does not commence till a whole rib and interspace beneath its usual position, that carried down below the ribs in the usual parasternal line it rests for rather more than a similar space upon a tumour whose pulsations are the only ones to be felt in that region, and whose systolic impulse is synchronous with the first sound audible in the mitral area, and occupies its appropriate rhythmical position in relation to the second sounds, aortic and pulmonary, which have their position of maximum intensity depressed in exact proportion to the apparent depression of the entire cardiac dulness, the conviction seems irresistible that the heart as a whole is displaced downwards. And this conviction gains strength when we find that the liver, notwithstanding its considerable enlargement, is also displaced downwards, and especially when we find that the condition of the lungs is such as efficiently to account for this downward displacement. For the pulmonary percussion note indicates the presence of an abnormal amount of air in the lungs, especially in their upper lobes; while the defective movement of expansion, the imperfect character of the inspiratory murmur, and the prolongation of the expiration, all indicate that this depends upon abnormal retention of the air within the lung and deficient elasticity of the pulmonary tissue, such as we know to be the result of that over-distension and rupture of the air-cells which is termed emphysema, uniformly associated with inelastic enlargement of the parts of the lung affected, and, if sufficiently extensive, equally uniformly

associated with depression of the heart and liver, and extension downwards, as in this case, of the area of pulmonary resonance. Further, we know that destruction of the walls of the air-cells must cause a permanent diminution of the area of the pulmonary capillaries, and consequently an irremediable obstruction to the circulation through the lungs, which must infallibly produce a permanently incurable dilatation of the right ventricle, and regurgitation through the tricuspid valve. Hence the physical signs in this case give a perfectly sufficient explanation of all the phenomena present, upon the only tenable supposition, viz., that here we have to do with emphysematous lungs producing depression of the heart and liver, enlargement of the right ventricle, regurgitation through the tricuspid valve, and secondary enlargement of the liver from venous congestion; while it cannot be denied that a thirty years' history of more or less permanent bronchitis is perfectly sufficient to account for the emphysema present, into the pathology of which we need not further enter at present.

This case is one of the best-marked instances of incurable tricuspid regurgitation I have ever seen, and with it I conclude the history of this lesion.

Careful attention to the phenomena described will enable any one readily enough to detect enlargement of the right ventricle, and to determine its amount and its seriousness as measured by that. And in this respect you will observe, in regard to the case last narrated, that though from its cause it was certainly incurable, yet the regurgitation was not very great in amount. And this we deduce from several facts: first, the right ventricle could be partially rehabilitated, an impure first sound was audible every third beat or so after treatment for a short time; and second, there was no evidence of any great venous remora, there was no venous pulsation in the neck, there was no venous pulsation in or communicated to the liver, there never was any trace of albumen in the urine, and there never was any dropsy. The prognosis in this



patient is not absolutely unfavourable; he is certainly incurable, but with care he may live a long time; his cardiac affection will undoubtedly kill him in the long run, but in itself it will take a good while to do so, and he is more likely to be cut off by some acute intercurrent pulmonary attack.

In relation to prognosis, then, you will remember that tricuspid regurgitation associated with, and depending solely upon, an acute attack of bronchitis, is a perfectly remediable affection, and frequently permanently curable. It is a measure of the amount of pulmonary congestion present, and therefore of the seriousness of the pulmonary disease, but in itself it is of little consequence.]

When associated with mitral disease, however, and especially as it so often is with mitral constriction, tricuspid regurgitation always renders the prognosis most grave. Even when the dilatation of the right side has been precipitated by an attack of acute bronchitis, its rehabilitation is difficult to produce, and is never more than temporary; the end is always hastened, and often rapidly so.

In connection with pulmonary emphysema, tricuspid regurgitation is equally incurable, but the prognosis as to the duration of life is not so grave. And it is not difficult to understand why this should be; for we know that the pernicious influence of tricuspid regurgitation is not to be estimated by the degree of venous stasis produced, but rather by the diminution of the aortic blood pressure to which it gives rise, and of which that venous stasis is a measure. But a limitation of the area of the pulmonary capillaries, even sufficient to induce tricuspid regurgitation, diminishes the amount of blood in the aortic system only by the amount of blood actually cut off. So long as that is consistent with the maintenance of life, no important change can take place for a very long time indeed without the supervision of secondary causes; nay, an improvement in the patient's condition is more likely to occur than a deterioration, because that must necessarily follow any partial rehabilitation of the right ventricle, or any



increase of function in the healthy parts of the lung, both of which are likely to take place with any improvement of the patient's health. Hence, tricuspid regurgitation arising from pulmonary causes, and compatible with life, may, though incurable, persist almost indefinitely, until the aortic blood pressure becomes fatally lowered by some secondary cause.

In connection with mitral constriction, however, the super-vention of tricuspid regurgitation has quite another and a much more important signification. Physical laws teach us that the force and velocity with which a fluid passes through any orifice are increased in a certain definite ratio by the amount of pressure brought to bear upon it. We can readily understand, therefore, that in cases of mitral stenosis the maintenance of the aortic blood pressure is greatly dependent upon the amount of hydrodynamic pressure existing within the pulmonary circuit. And we can also equally readily understand that whenever the intrapulmonary blood pressure is diminished by any efficient cause, such as by the occurrence of tricuspid regurgitation, the immediate result is a diminution of the flow through the stenotic orifice, and a corresponding diminution of the amount of blood thrown into the aorta,—the amount of blood sent forwards being reduced with a rapidity and in a degree commensurate with the influence of the pre-existing hydrodynamic pressure in maintaining it, the reduction having a strong tendency to be permanent, and to increase at a rate proportionate to the degree of stenosis and amount of tricuspid regurgitation present.

In tricuspid regurgitation, therefore, depending upon pulmonary obstruction, the amount of influence exerted in diminishing the quantity of blood sent forward, and thereby all the secretions and other vital functions, is to be estimated merely by the amount of blood cut off. But in tricuspid regurgitation depending primarily on mitral stenosis, the amount of influence exerted in diminishing the amount of blood thrown into the aorta is to be measured not merely by the ratio between the stenotic and the normal auriculo-

ventricular orifice, but by that, plus the diminution of the hydrodynamic pressure within the pulmonary circuit. There can, therefore, be no difficulty in understanding which is the most serious lesion, nor why it should be so, both being equally permanent. Hence, in tricuspid regurgitation following emphysema, the patient may maintain a moderate degree of health for a long time, though he will never be robust, and will always be placed in extreme danger by any attack of acute pulmonary congestion.

In the treatment of tricuspid regurgitation, besides whatever subsidiary remedies may be employed, we must never omit the use of digitalis, remembering that the action of this drug is not merely to increase the force of the heart's action, but also the tone of the ventricular muscular fibres, and thus it promotes the contraction of the ventricular cavity, and when possible produces the rehabilitation of the tricuspid valve.

Even in cases of tricuspid obstruction, of which, as you are aware, I have had but little experience, I see no reason for withholding digitalis. In both of the cases of this affection which I have seen, this drug seemed to do good service. And as its tonic effect is exerted on the auricles as well as on the ventricles, and as increased energy of the auricular contraction is of the utmost importance in these cases, there is good reason for employing this drug in these cases. Nay more, digitalis acts undoubtedly on the muscular fibres of the circulating system generally, arteries and veins, and there seems every reason for believing that in appropriate doses it increases the energy of that systolic wave which passes over the whole circulating system, upon which the maintenance of the circulation in many of the lower animals so entirely depends, and which is certainly of some importance in maintaining the circulation even in the highest mammals.

## LECTURE VIII.

ON THE MURMURS AUDIBLE IN THE PULMONARY REGION, THEIR  
VARIETIES, AND DIAGNOSTIC SIGNIFICATION.

GENTLEMEN,—The so-called pulmonary area, the space between the second and third rib to the left of the sternum,\* has been not inaptly termed the region of romance, because of the various interpretations which have been given to the murmurs having their position of maximum intensity in that situation. Interpretations which have been only too apt to vary with the views and predilections of the commentators; and yet if there be any truth whatever in physical diagnosis, there can be no room for mere opinion in the interpretation of a murmur, whatever its situation, as that must rest upon physical, and therefore incontrovertible facts. The correctness therefore, of any diagnosis must depend entirely upon the ability of the observer to collect the necessary facts, and not upon any ripeness of experience, and the time is rapidly approaching when the knowledge of medical physics shall be so widespread as effectually to eliminate mere opinion from the diagnosis of diseases of the heart, an organ the condition of which we can ascertain in so many different ways, that with sufficient care we can be quite as certain of the state of its orifices at least during life as if we had it on the dissecting table.

Systolic murmurs, duly recognised as such by their coin-

\* *Vide* Lecture I., p. 29.



cidence with the apex beat or the carotid artery, and most distinctly audible between the second and third ribs to the left of the sternum, will be found to have two positions of maximum intensity. In one series of such murmurs this position of maximum intensity is not close to the sternum, though the murmur is also audible there, but an inch or more to the left of the left edge of that bone—just where we know that the appendix of the left auricle comes up from behind to the left of the pulmonary artery. When much dilated, the auricle extends still further to the left as well as downwards, and occasionally partly covers the pulmonary artery, yet even in its most dilated conditions leaves a space between the stethoscope, resting on the position of maximum intensity of the murmur, and the left edge of the sternum, equal to at least the breadth of the tip of the middle finger. In these cases this murmur, which is so commonly reckoned a pulmonary one, really depends upon regurgitation through the mitral valve, as was first pointed out by Naunyn.\* A systolic murmur audible in this position is a very common one, and in by far the larger proportion of cases, depends upon simple dilatation of the left ventricle of a curable character, is generally associated with a venous hum and with other signs and symptoms of spanæmia, and commonly passes for a hæmic murmur. I have already told you my reasons for adopting with Naunyn's interpretation of this murmur as explanatory of such cases, and pointed out the means of diagnosing them and of curing them.† In a much smaller proportion of cases, but still not infrequently, a systolic murmur with its position of maximum intensity in this situation, is due to the regurgitation almost always present in mitral constriction,‡ many circumstances favouring in these cases the con-

\* *Loc. cit.*, *Berliner klinische Wochenschrift*, 1868, No. 17, s. 189.

† Lecture VI., *passim*.

‡ I have introduced the word "almost" in this connection, because there are some few funnel-shaped valves in which it seems impossible to suppose that regurgitation takes place at all, and at all events it can only occur with difficulty and to a small extent.

vection of this murmur upwards to what we may term the auricular area, rather than its mere conduction downwards into what is termed the mitral area. These being the great dilatation of the appendix auriculi, and the concomitant tendency of the left apex to become depressed within the chest by the distended right ventricle, which in all these cases is usually more or less dilated, while to these favouring circumstances we must add the natural tendency of all murmurs to accompany the fluid veins which produce them, and to some extent also the original size of the appendix which varies in every individual. Possibly also the physical constitution of the murmur itself, and certainly the physical constitution of that portion of lung overlying the apex. All these circumstances coincide in favouring the communication of the soniferous impulses which cause the murmur to the walls of the chest, and therefore to our ear in the auricular area rather than at the cardiac apex.\*

In another series of such murmurs the position of maximum intensity is strictly limited to the true pulmonary area, viz., the second interspace close to the left edge of the sternum, right over the pulmonary artery, which in this region is normally so placed that one-half of its lumen is covered by the sternum, the other half lying to the left of that bone, and this series again falls to be subdivided into two categories, one associated with a perfectly normal condition of the pulmonary artery, and the other with an abnormal condition of that vessel.

\* In his recently published work on *Diseases of the Heart and Aorta*, Dublin, 1875, Dr Hayden, in reference to these views, says, at p. 1001, that a presystolic murmur is rarely audible in the second interspace, and when audible there may be readily recognised by its relation to the first sound and the cardiac impulse. In this Dr Hayden is no doubt correct enough, only it has nothing to do with the occurrence of a systolic murmur in the region referred to. When he also says that "a murmur of mitral reflux should be readily distinguishable by its special localisation and diffusion," I also perfectly agree with him, only Naunyn has shown that there are many facts and many physical circumstances which prove that the localisation and diffusion of a murmur of mitral reflux are not necessarily limited to the mitral area, but are very frequently to be found in the second interspace, and with cases of this nature it would seem that Dr Hayden has not yet made himself familiar.

I shall consider first the one set of cases and then the other, pointing out the diagnostic phenomena peculiar to each, and their signification.

In the whole course of my experience of cardiac disease, I have met with nothing more extraordinary, nor at first sight more inexplicable, than the coexistence of a loud systolic murmur in the pulmonary region with a perfectly normal heart. Such cases do, however, occur, and they simulate aneurism of the sinus of Valsalva so closely in the roughness of the systolic murmur, the distinctness of the second sound, and the existence of abnormal pulsation, as to be somewhat puzzling, and apt to mislead. The only two points in which they differ from aneurism in this situation are—first, in the entire absence of pain, which we know does not always exist even though aneurism be present; and second, in the feebleness of the pulsation compared with that of the heart, a sign which though of undoubted value in the diagnosis of aneurism, is not wholly nor at any time solely to be relied upon as of excluding the idea of aneurism.

Dr H. Quincke of Berlin has recently\* devoted his attention to the subject of these murmurs, and as in many respects his observations coincide with my own, I shall first give his explanation of these inorganic pulmonary murmurs before I relate my own cases. But first I may tell you that Dr Quincke has pointed out, and related one case in proof, that in certain rare cases of mitral stenosis in which the right ventricle is hypertrophied but not dilated, while the pulmonary artery is dilated, a murmur may arise at the orifice of the pulmonary artery from the formation of fluid veins there, as the blood passes through the comparatively narrow healthy opening into the dilated artery beyond. Physically this is a possible cause of a systolic murmur in this situation, though it must certainly be a very rare one. In the case in point

\* Beiträge zur Entstehung der Herztöne und Herzgeräusche. *Berliner klinische Wochenschrift*, 1870, No. 21, s. 249. *Vide also Ed. Med. Journal*, Jan. 1871, p. 667.



the appendix auriculi was thrombosed and lay deep in the chest; the auricular origin of the murmur in this case from convection was therefore precluded. But he has also pointed out that there is a series of cases, of which he has related six, in which the systolic pulmonary murmur is accompanied by an uncovering of the base of the heart by retraction of the left lung from various causes, and he supposes that in these cases the systolic murmur is produced by compression of the pulmonary artery by the heart during its systole. The murmur being caused by the passage of the blood, through the compressed and narrowed portion of the artery, into the uncompressed and therefore comparatively dilated portion beyond, a state of matters which must be accompanied by the production of fluid veins at the part artificially narrowed, and which, therefore, if it exist, must be regarded as an efficient physical cause for the production of a systolic murmur. He has based his views as to the possibility of the production of this artificial narrowing, first, upon that fact that the pulmonary artery could in all these cases be felt and seen to pulsate, this pulsation being accompanied by a localised systolic murmur, which ceased to be audible whenever the pulmonary artery again became covered by lung, and its pulsations were no longer perceptible by touch or sight; and second, because although the reason why the lungs do not overlap the heart in these cases is not always quite plain, yet in most of them there was sufficient cause present to account for the imperfect expansion of the lung. In one case the cause was pregnancy, the murmur disappearing on the occurrence of abortion, in another pneumonia, and in the others debility from various causes. He has also shown that so long as the imperfect pulmonary expansion lasted, just so long the murmur continued, and so soon as a more perfect expansion occurred, then the murmur ceased; and on measuring the amount of expansion of the chest in both conditions, he found that after the disappearance of the murmur the chest expansion was greater by a few centimetres than formerly, and what is more to the point,

that the absolute cardiac dulness was also diminished *pari passu* with the return of the normal lung expansion, and the disappearance of the pulmonary bruit. Moreover, in many of these cases forcible expansion of the lungs is sufficient to cause complete cessation of the murmur, for just so long as the breath can be held. He has also anticipated certain objections to his theory, due to the fact that a similar systolic murmur is not always audible even when pulsation of the pulmonary artery is visible, by pointing out first, that murmurs are very often absent, even when all the conditions necessary for their production are present, as is so frequently the case in mitral stenosis; and second, that in other cases any compression of the pulmonary artery great enough to produce a murmur, may be prevented by the peculiar formation of the chest, and especially by any considerable prominence of the cartilages of the second and third ribs.

You all know very well that compression of any artery, if it be capable of materially narrowing its calibre, is, and from physical causes must be, sufficient to produce a systolic murmur at the part compressed, provided the blood flows through it with sufficient force; if we can therefore connect retraction of the left lung and uncovering of the heart with a systolic pulmonary murmur, which disappears when the heart is more fully covered by deep inspiration or otherwise, which disappears even while that retraction persists, whenever compression of the pulmonary artery by the cardiac systole is by some cause or other precluded, there being at the same time no other apparent source of cardiac murmur present, then I think you cannot but agree that we have the very strongest *à priori* reasons for accepting Quincke's explanation as correct.\* In

\* Quincke supposes that in the normal condition the ventricular systole merely forces aside the overlapping edge of the left lung; when that is done to its hand by retraction of that lung, this portion of the systole is expended in flattening the pulmonary artery. Hayden, *op. cit.*, p. 1002, argues that if this were true, then in every case of visible pulsation of the pulmonary artery we ought to have a murmur. Quincke has already sufficiently disposed of this argument. Hayden also very properly points out that in many cases of mitral



the cases which follow you will find all these postulates present and whatever difficulties may lie in the way of accepting Quincke's explanation, it seems still more difficult to explain the occurrence of such murmurs in any other way consistent with the physical laws as to the production of sound.

CASE XX.—William M'Leod, aged 32, formerly a soldier now a van-driver, was admitted into Ward V. of the Edinburgh Royal Infirmary on 20th May 1870. He complained of an occasional beating in the left breast, accompanied by great breathlessness, apt to be brought on and increased by exertion. While in India as a soldier some years previously, he had been laid up for a month with what appeared to have been, from his description, an attack of rheumatic fever; he also had syphilis there about fourteen years ago. His pupils were natural; his radial pulses slightly unequal, the left being a shade smaller than the right; the humeral pulses did not, however, vary. On percussing the cardiac region transversely in the nipple line (along the upper edge of the fourth rib), dulness was found to extend for about one inch and a half from the left edge of the sternum to within half an inch of the nipple; on percussing longitudinally from the clavicle downwards one inch from the left edge of the sternum, dulness was found to commence at the superior margin of the second rib, and to extend down to the liver dulness (left lobe). The apex beat was between the fifth and sixth ribs and just below the nipple. Between the second and third ribs on the left side the dulness extended for rather more than an inch from the left edge of the sternum, and within this space pulsation was to be felt—less forcible, however, than that of the heart. On auscultating over the apex beat, a loud rough murmur was heard just preceding the apex beat, up to which it ran—a presystolic murmur occupy-

stenosis we have pulsation in this region unaccompanied by a murmur, and this pulsation he supposes to be due to dilatation and distention of the pulmonary artery, but the position of the impulse in such cases is quite to the left of the pulmonary artery, and could only be produced by a large pulmonary aneurism. After death we find in such cases a dilated auricle, but never any pulmonary aneurism.



ing the time of the auricular contraction ; the first sound was not pure, but there was no systolic murmur audible in this area ; the heart's action was regular. Between the second and third ribs on the left side a loud and somewhat rough systolic murmur was audible, closed by an accentuated second sound ; this murmur was propagated down to the fourth rib on the left side, and also across the sternum to the space between the second and third ribs on the right side, where the aortic valves were heard to close with decidedly less force, comparatively. The systolic bruit became always very faint, and occasionally inaudible, on the patient assuming the erect posture, the presystolic bruit remaining unchanged. The diagnosis was mitral stenosis, and probably aneurism of the ascending portion of the aorta, arising just above the valves ; the latter portion of the diagnosis being based on the existence of a pulsating tumour, with dulness, between the second and third ribs on the left side, over which a loud rough systolic bruit was audible followed by an accentuation of the second sound in that region, much greater than that usually heard in cases of simple mitral contraction. There was no reason to suppose that the systolic bruit was of mitral origin, propagated upwards by convection ; the possibility of this was duly considered, and rejected because of the entire absence of any such murmur in its usual position,—(this, however, is an error, as we now know that a mitral systolic murmur is not infrequently audible in the auricular—pulmonary—area even though inaudible in the mitral area),—and also because of its exceedingly loud and rough character, and the wide area of propagation ; the “probably” was added because so many similar cases had been seen of late, that it appeared either that aneurism in this situation was more common than is usually supposed, or there was some fallacy in the diagnostic signs, and I was inclined to hold the latter opinion. He was discharged greatly improved, on the 20th of July. Previous to his discharge he was seen by Professor Sanders, who confirmed the diagnosis especially as to the possibility of its being a case

of aneurism. On the 16th of October he was readmitted, complaining of irregular attacks of ague, which he had formerly suffered from in India; he had been taking arsenic for a week, in full doses, without benefit. He was ordered five grains of quinine every two hours, and under this treatment he rapidly improved. On the evening of the 27th of October he had a rigor followed by fever, and a restless night; on the 28th he was feverish, with quick respirations, rusty gelatinous sputum, fine crepitation over right lung posteriorly; no dullness. On the 29th he was much feebler, respirations 60, otherwise as yesterday; and in spite of free stimulation he died at 3 P.M.

*Autopsy, 1st Nov.*—On opening the thorax the left lung was seen to be retracted upwards and backwards, receding from the mesial line opposite the second and third ribs; it dipped down opposite the fourth and fifth ribs, leaving uncovered the whole of the right ventricle, the apex and lower half of the left ventricle, and to a slight extent the pulmonary artery also. Amount of serum in thoracic cavity normal. The heart weighed  $19\frac{1}{2}$  ounces, and was hypertrophied and slightly fatty. The pulmonary and tricuspid valves were healthy. The cusps of the aortic valves thickened, and slightly incompetent; the mitral valve much thickened especially its aortic segment. The auriculo-ventricular orifice on the left side was so contracted as only to admit the point of the little finger. There was some cretaceous deposit at the junction of the auricle and the ventricle; the cordæ tendinæ of the mitral valve thickened; the aorta healthy. Both lungs were deeply congested, and floated heavily—especially the right one—but neither were properly speaking hepatised. The liver weighed 4 lbs., and presented an indistinct nutmeg character; its capsule was healthy. The kidneys were pale and fatty, the cortical part increased one-third in volume; the capsules free, the surface beneath smooth. The spleen weighed 2 lbs.  $1\frac{1}{2}$  oz.; it was softened, and on section presented a few hæmorrhagic spots, varying from the size of a shilling downwards.



The autopsy, while thus confirming the diagnosis in every other point, gave no support whatever to the idea of an aneurism, not a trace of this having been observed; but for this I was fully prepared by the previous occurrence of the following still more remarkable case:—

CASE XXI.—James Morrison, aged 31, a bricklayer, admitted to Ward V. on 25th April 1870, complaining of soreness in the chest, headache, dyspnoea, and some swelling of the face and body generally. I shall omit the general history of the case, which was that of an ordinary case of Bright's disease, merely premising that, though by no means anæmic, he was of a somewhat sallow complexion; and that the dropsy present was only an inconsiderable amount of general anasarca, depending upon an early stage of cirrhosis of the kidney, with slight traces of amyloid degeneration, as ascertained by inspection after death. He was a powerfully-built man, with all his other organs and functions in perfect health and normal, with the exception about to be mentioned. His pupils were both equal, and both pulses equal at the wrist. The apex of his heart beat between the fifth and sixth ribs, just outside the nipple line, and was somewhat diffuse. Cardiac dulness extended longitudinally one inch to the left of the sternum from the lower edge of the second rib to the liver dulness, and transversely in the nipple line (along the upper border of the fourth rib), from the left edge of the sternum to the left nipple, a distance of three inches. Between the second and third ribs, on the left side, there was an evident pulsation, which was most distinctly perceived on deep expiration. This pulsation extended for three-quarters of an inch from the edge of the sternum, and ended abruptly; dulness in this situation, however, extended to the left for quite two inches, but scarcely encroached at all upon the sternum. Upon auscultation, the first sound over the apex was rather feeble, but distinct and without bruit; the second sound was somewhat accentuated. At the right edge of the sternum, between the second and third ribs, the first sound was still heard, somewhat feeble,



but free of murmur, while the second was more markedly accentuated, and this accentuation increased as the stethoscope was moved across the sternum till it attained its maximum over the dull pulsating tumour already described; and here—that is, between the second and third ribs on the left side—it was preceded by a specially loud and rough systolic bruit, very well marked and distinct, and presenting none of the usual characteristics of a so-called hæmic murmur. A trace of this bruit could be heard over the innominate and right carotid arteries, but not even a trace was audible over any of the other vessels, though the accentuation of the second sound was more or less distinctly heard in both subclavian and in both carotid arteries. It is impossible to conceive any objective symptoms which could more closely resemble those of an aneurism arising from one of the sinuses of Valsalva and passing to the left, with this only proviso, that the pulsation was fainter than the apex beat; and this cannot be, under all circumstances, accepted as efficient negative evidence. Subjective symptoms, relating to either aneurism or cardiac disease, were entirely absent; their absence, however, is certainly not efficient evidence of the non-existence of an aneurism, though it would have thrown a halo of suspicion around any case with symptoms less well marked than this one appeared to possess.

This patient was unfortunately seized, while under treatment, with an acute attack of erysipelas of the head, of which he died upon the 29th of June. At the autopsy on the 1st July, the left lung was found to be somewhat retracted, leaving the upper part of the heart and pulmonary artery rather more uncovered than usual; the aorta was found to be very slightly dilated, but the heart itself was, with the exception of a slight enlargement of the left ventricle, normal in every respect; the pulmonary artery was perfectly normal and its valves healthy. The interior of both the aorta and pulmonary artery, and their chief branches, were perfectly smooth, and wholly free from every trace of atheroma.

CASE XXII.\*—J. W., aged twenty-eight, a fish salesman, admitted June 18, 1874, to bed 7, Ward V., of the Edinburgh Royal Infirmary, complaining of lassitude, weakness, and pain in the abdomen. About six months previously he had pain in the chest, severe cough, night-sweats, and loss of appetite, brought on by exposure to cold and wet. Under treatment these symptoms in a great measure disappeared, but he has always since that time had some cough, and has never regained his strength. About a month before admission he first noticed a swelling in his right side, which was painless in itself, but accompanied by pain in the stomach after every meal, and occasionally at other times. The patient has always been healthy, except that in 1869 he passed through an attack of cholera, and about a year subsequently he had a fever, nature unknown. His family history is good; his father and mother, three brothers, and two sisters are all alive, and in good health. The patient states that he has never been abroad, has never suffered from ague, rheumatism, or syphilis, and has never had any suppurating wound or sore, except an occasional trifling scratch; but he has been in the habit of drinking beer and other malt liquors in large quantities. On first admission he was fairly developed as to muscle, but his face was thin and his expression somewhat anxious. His temperature was 99°, his respiration 16 per minute. He had night-sweats, and occasional cough, especially at night. The hepatic region was found to be tumid but soft, and there was considerable pain on pressure just above the umbilicus.

On July 3 the patient had emaciated somewhat, his muscles were soft and flabby, sweating at night continued. Since his admission his temperature had varied from 97° to 104° in a most irregular and capricious manner, being only once 104° (on the evening of June 26), and 98° only on the preceding and following mornings, the average temperature being little over 100°; his respirations also varied, but averaged about 20 per minute. He still coughed severely at times, chiefly during

\* Reported by W. R. Gibson, clinical clerk.



the night. The thorax was well formed, and expanded apparently equally on both sides. Lung-percussion was normal. On auscultation rough vesicular breathing was heard over both lungs, both in front and behind, mingled with an occasional mucous rattle or a rhonchus. The pulse was of fair strength, 90 per minute; peripheral arteries and veins normal. There was no cardiac pain or palpitation complained of, but on inspection of the cardiac area the heart's pulsations were more apparent than usual, while between the second and third ribs on the left side, close to the edge of the sternum, distinct pulsation was observed, which seemed to propagate itself upwards to the root of the neck as a slight tremor running over the superficial parietes. At one inch from the left edge of the sternum dulness commenced at the lower border of the second rib and extended downwards to the liver dulness; in the intermamillary line dulness commenced at the right edge of the sternum and extended across to the left for a distance of four inches; between the second and third ribs dulness extended to the left of the left edge of the sternum for a distance of two inches and a half. In the mitral, tricuspid, and aortic areas the normal heart-sounds were distinctly audible; in the pulmonary area the first sound was entirely replaced by a loud, rough murmur, followed by a more than usually distinct but not accentuated second sound.\* This systolic pulmonary murmur was found to disappear completely when the patient took a deep inspiration and held his breath, reappearing at once on expiration; it was not propagated in any direction. The tongue had a white coating, with a triangular red part in the centre, the apex of which coincided with that of the organ. The patient's appetite was impaired, with a feeling of oppression over the scrobiculus cordis after meals, and much thirst. The bowels were irregular; most frequently loose. The abdomen appeared distended, and had a peculiar tense elastic feeling all over it; pressure on any part revealed slight tenderness, which culminated in positive pain at a point just above the umbilicus

\* The element of tension being wanting.—*Vide* p. 30.



in the mesial line. Round the umbilicus the patient measured thirty-one inches and a half; three inches higher he measured thirty-three inches and a half, this increase being markedly on the right side, and due to bulging of the liver, which was enlarged, the liver dulness in the mammary line measuring seven inches. This organ was soft and elastic to the touch, and slightly tender on pressure. The splenic dulness was somewhat increased. Micturition was more frequent than usual, and occasionally accompanied by pain along the course of the urethra. The urine was of a dark straw colour, with a large deposit of mucus; its specific gravity was 1015; it was acid, and averaged in quantity from fifty to sixty ounces, and contained a large quantity of albumen, but no sugar or bile. On microscopical examination, the deposit was found chiefly to consist of amorphous matter, with epithelium, and a few fragments of hyaline and slightly granular casts.

The subsequent history of the case was that of gradual emaciation and sinking, with an irregular temperature averaging about 100°; falling sometimes to 97°, and again rising to 102°; a persistence and gradual increase of the diarrhoea: a discharge of highly albuminous urine—never below thirty-five ounces, and sometimes as high as ninety ounces; a gradual but trifling accumulation of fluid in the abdomen and in the cellular tissue of the legs and feet; and latterly the development of a very slight degree of jaundice.

On July 13 it is noted that the painful spot above the umbilicus had become exceedingly tender to touch; nothing was to be felt there, but low down in the right iliac region a small limited hard patch was to be felt at the lower border of the liver.

On July 21 the systolic pulmonary murmur had entirely disappeared, and was not again heard.

The patient gradually sank and died on July 30.

*Autopsy.*—Body much emaciated; skin and conjunctivæ somewhat jaundiced. There were pleuritic adhesions on the left side extending down the whole thorax at the junction of the

ribs with the cartilages. The fibrous sac of the pericardium was more fully exposed than usual, owing, apparently, to the lungs having collapsed very markedly when the chest was opened. Heart was of small size, weighing seven ounces. All its chambers contained decolorised blood-clot. On the anterior surface of the right ventricle there was a milk spot, which measured an inch and a half in length and three-quarters of an inch in breadth. The thickening of the pericardium at this spot was unusually marked. The chambers of the heart were all of the natural dimensions. The tricuspid and mitral valves were quite natural. The tricuspid orifice admitted four fingers; the mitral three. The aortic and pulmonary valves were competent and otherwise quite natural. A tough decolorised blood-clot occupied the pulmonary artery, and extended along its primary ramifications in both lungs. The wall of the aorta was free from atheroma and otherwise quite natural. On microscopical examination the muscular fibres of the heart were found to be quite healthy. Lungs: The left weighed fourteen ounces, the right fourteen ounces and a half; both were somewhat congested in their posterior three-fourths, and there was a little emphysema at the anterior edges of both. Abdomen: The peritoneal cavity contained rather more than two quarts of turbid serum, in which floated numerous flakes of soft recent lymph. Liver weighed six pounds one ounce and a half; its surface was smooth, but its capsule presented ramifying lines of white opacity, and in the upper surface of the right lobe there was an opaque thickening with puckered stellate edges like a cicatrix. The thickening, however, did not extend beyond the capsular surface. On section the organ presented a slightly granular aspect, and felt exceedingly tough and fibrous. It also presented to the naked eye some of the characteristic appearances of the waxy degeneration, and it gave the usual staining on the application of the iodine test, but not very markedly. On microscopical examination a very marked increase of connective tissue was observed both within the lobules and at their peripheries. Pancreas



was of natural size and healthy texture ; it weighed, with a few lymphatic glands attached to it, four ounces and a half. Close above the margin of its head and upper part of body there were a great number of enlarged lymphatic glands, varying in size from that of a pea to almost that of a pigeon's egg. From the pancreas a string of these glands extended up to the liver between the folds of the gastro-hepatic omentum. The mesenteric lymphatics and the prevertebral lymphatics around the coeliac axis, and for two inches and a half below, were enlarged, and had undergone the same degeneration as those above the pancreas. On microscopical examination of the glands the lymph cells were found to have run together into waxy translucent clumps, which, as well as the vessels, took on the waxy reaction very markedly. There was no obstruction of the cystic or hepatic ducts. The spleen was much enlarged, and weighed thirteen ounces and a half. Its capsule was firm and fibrous, and the Malpighian bodies were unnaturally prominent. On using the iodine test the Malpighian bodies exhibited the staining very markedly. The kidneys were both enlarged ; the right weighed eight ounces, the left seven ounces. The cortical substance was much increased, and the organs were very much congested. The Malpighian corpuscles exhibited the staining with iodine. Intestines : The mucous membrane presented the waxy reaction very markedly.

Now, you will observe that all these cases agree in this, there was in every one of them retraction of the left lung, and uncovering of the heart and pulmonary artery, the exposure of the latter permitting its pulsation to be readily seen and felt, a pulsation which could not possibly be mistaken for that of a dilated auricle, inasmuch as it occupied a very different position, close to the edge of the sternum, and ended abruptly just when we got to the auricular area. Moreover, the finger laid upon the pulsating part could distinctly feel in every case the click of the valves, so that its arterial origin could not possibly be mistaken even had the left auricle been coetane-



ously dilated, which it was not in any case except the first, and even in it not to any great extent. The uncovering of the pulmonary artery also brings the valves nearer the surface; hence their closure is not only more easily felt, but also more easily heard; hence the appearance of accentuation which it assumes even when it is not actually accentuated from other causes. In the second case narrated there was slight dilatation of the aorta, too limited to be made out by percussion, but yet sufficient, no doubt, to produce both accentuation of the aortic second, and also a systolic murmur, propagated into the arteries of the neck, and this fact, coupled with the greatly increased intensity of the second sound over the pulsating tumour to the left, as well as the peculiar localised roughness of the murmur which preceded it, complicated the case, and made it long to be regarded as an aneurism of one of the sinuses of Valsalva. Indeed, the very peculiar loudness, roughness, and localised character—not propagated in any direction—of the murmur make it in every respect one of the most remarkable in the history of cardiac diseases. In the cases described there is little reason to doubt that the spanaemia present in all of them assisted in the causation of the murmur. You will find it noted in regard to the first case that the murmur disappeared whenever the patient assumed the erect position, possibly because in this position the necessary compression of the pulmonary artery was prevented by the heart's weight dragging it down. In the third case, the systolic murmur in the pulmonary artery disappeared entirely whenever the patient took a deep inspiration, and held his breath, reappearing at once on expiration. Moreover, nine days before the patient's death, when the heart must have been already somewhat atrophied and certainly enfeebled, and therefore not only less able to compress the pulmonary artery, but less able to drive the blood through it with a force sufficient to produce a murmur, then this murmur ceased for ever. It seems impossible to account for the retraction of the lung in the two first cases; in the third one we had two

efficient causes contributing to prevent a complete covering of the heart during ordinary inspiration: first, we had the greatly enlarged liver and the pain in the abdomen, and then we had the string of adhesions along the junction of the ribs with the cartilages on the left side, which, though not tight enough to prevent the covering of the heart to its normal extent on forced inspiration, yet, no doubt, contributed their quota in preventing full covering in the ordinarily restrained respiration of this patient. In a fourth case, of which unfortunately I cannot give you full details, the cause of the retraction of the lung was only too evident. The patient was, under my care for some time in Ward XIII., presenting all the ordinary phenomena of pulmonary phthisis, affecting mainly the upper lobe of the left lung, and at that time she had no cardiac murmur whatever. She improved considerably, and was discharged. About a year after she was admitted into Ward XV., under the care of my colleague in the Infirmary, Dr Haldane, and he directed my attention to it as a remarkable case of pulmonary bruit. In this case the upper lobe had several small cavities in it which had cicatrised and contracted, this contraction of the lung completely uncovering the base of the heart, and *pari passu* with the retraction of the lung from the base of the heart we had developed a loud, rough, localised murmur in the pulmonary artery. Now, though these cases cannot be held to have proved the correctness of Quincke's theory, they yet lend a very strong support to it, and it will be very difficult to propound another, equally simple and probable, which will harmonise so well with all the facts associated with this localised bruit as we find them in nature.

The class of systolic pulmonary murmur associated with an abnormal pulmonary artery need not detain us long. These cases are always congenital, and always rare; they are more objects of medical curiosity than of medical treatment, and each individual case presents, besides the ever present systolic murmur in the pulmonary artery, other murmurs and signs of coexisting congenital malformation, which vary somewhat in



each case, and I cannot conclude this lecture better than by giving a full account of the only case of the kind which has come before me during my connection with this Infirmary.

CASE XXIII.\*—Anne C——, aged nineteen, a dressmaker, born in Aberdeen, and residing at Leith, was admitted to bed 10, Ward XIII., on December 1, 1873, complaining of great pain over the præcordia, extending to the left back and shooting down the left arm, and of a bad sore throat. The patient is usually deeply cyanotic, but varies in complexion; the digits on all four extremities are clubbed in a very marked degree and the nails aduncated.

Her mother stated that last February (1873), after receiving a fright, her daughter was seized with a sudden pain in the left side, which finally settled over the præcordia. At that time she suffered much from palpitation and general feeling of oppression in the chest. This state of matters lasted till May. After a short period of comparative health she was, in June, seized with a shivering fit, and shortly after coughed up two mouthfuls of dark blood, and several more of a lighter colour. Since then she has always been ailing. Her mother also stated that ever since birth she had been of a blue colour, specially marked after any exertion and in cold weather. Shortly after her birth her father contracted syphilis, which he communicated to her mother, and, through her mother, to herself and to all the children born subsequently. So that, while her brothers and sisters born previously are healthy, she and her mother both suffer from acquired syphilis, and those born subsequently from congenital syphilis.

Anne C—— is fairly developed, rather above the average height; and weighs 8 st. 3 lbs. Her facial expression is dull, her complexion is of a rosy hue, sometimes livid; her lips generally, and her clubbed finger-tips always, livid, and the same remark may be made of her ankles and toes; her skin generally is not discoloured, is sometimes warm, moist, and clammy, at others cold and dry; temperature 98·4°. Her

\* Case reported by Mr Kelly, clinical clerk.



pulse varies from 68 to 75 per minute, is feeble, and equal at both wrists. The jugular veins are small, but distinct pulsation is visible in them. She has dyspnoea on the slightest exertion, no palpitation, but severe pain over the præcordia. On inspection the chest appears normal; no pulsation anywhere visible. On palpation no impulse is anywhere perceptible, even when she leans forward, but a considerable thrill is to be felt over the base of the heart. On percussion from above downwards, one inch from the left of the sternum, cardiac dulness begins at the level of the upper edge of the third rib, and extends down to the liver dulness. At the level of the fourth rib dulness begins three-quarters of an inch to the right of the sternum, and extends to the left for a distance of four inches and three-quarters. The aortic dulness is found to extend upwards to within one inch of the top of the sternum. On auscultation a loud systolic murmur is distinctly audible over the whole cardiac area, propagated with more distinctness towards the left than towards the right axilla. By careful auscultation posteriorly a faint systolic murmur is audible, evidently due to propagation through the bones. In the mitral area (between the fifth and sixth ribs, two inches and a half to the left of the sternum) this systolic murmur is shrill and distinct, followed by a faint second sound. In the tricuspid area (sternal end of the fourth, fifth, and sixth ribs, on the left side) the systolic murmur is louder, more prolonged, and followed by a more distinct second sound. In the aortic area (sternal end of the second rib on the right side) the systolic murmur is shrill, distinct, and followed by a distinct second sound, often markedly accentuated, both sounds being propagated into the carotids. In the pulmonary area (sternal end of the third rib on the left side) the systolic murmur is louder and rougher than in any other part of the cardiac area, and followed by a second sound which, though always distinct, is at times quite accentuated; when it is most distinct the aortic accentuation seems to be least marked. Her respirations are 20 per minute; dyspnoea on the slightest exertion; pain in

the chest; no cough or expectoration; voice hoarse; pulmonary physical signs normal. With the exception of sleeplessness, occasional shooting pain over the left temple, and the præcordial pain already referred to, her nervous system is normal. There is considerable difficulty of swallowing, from syphilitic ulceration of the uvula and swollen and congested tonsils, also slight atonic dyspepsia, digestive system otherwise normal. The urine is of a pale sherry colour, acid, sp. gr. 1020, containing a small amount of albumen; otherwise normal; no deposit. Menstruation regular.

This history, thus concisely narrated, contains within itself all the elements for an accurate diagnosis. The history and symptoms of acquired syphilis, though sufficiently distressing in themselves, are purely episodic in relation to the more serious affection under which this patient labours—an affection which, from its congenital character, has necessarily embittered all her life, and must shorten her days. The cyanotic condition of her surface, which has prevailed from birth, and the marked clubbing of all her digital extremities, point with unfailing accuracy to the existence of some congenital central lesion of her circulation. With regard to the production of cyanosis, three views are prevalent: first, there is the theory that it depends upon intermixture of the arterial and venous blood currents; second, that it is caused by venous congestion, depending upon obstruction to the onward flow of the blood current from whatever cause; and, third, that it arises from a combination of the two preceding causes. Clinical experience, however, teaches us that even where the obstruction is very great, as in extreme constriction of the mitral opening,—such as one now before me, which though somewhat enlarged by long maceration in spirit, only measures five millimetres by eight—the cyanosis is never so great as in those cases where, from congenital defect, intermixture of the two kinds of blood is possible. Whatever, therefore, may be the explanation,—and it is a subject still open to inquiry,—there can be no doubt that, though the absence of great cyanosis does not



disprove the structural possibility of intermixture of the blood currents, its presence is a certain proof of its occurrence, and therefore of the presence of those malformations which render it possible. This is confirmed in the present case by the existence of marked clubbing of the digital extremities—a condition which must have a cardiac origin if it extend, as in this case, to all four extremities in any marked degree. The presence of these conditions, therefore, enables us to conclude with certainty that in this case we have to do with congenital cardiac malformation, involving some condition capable of permitting intermixture of the arterial and venous blood currents. Now, apart from congenital deficiencies of the septa alone, which to any considerable extent are extremely rare, cause little if any cyanosis, and either give rise to no murmurs or to murmurs quite different in character and propagation from those in this case, we learn from the researches of those pathologists who have studied these malformations, that the probability of the original lesion being in the pulmonary artery increases with the age of the sufferer over fifteen. The age of our patient being nineteen, there is therefore a very strong probability that her primary lesion has been in the pulmonary artery, and the marked rough systolic murmur heard in the pulmonary area, and propagated with most distinctness towards the left axilla, renders this certain. The distinct and accurate closure of the pulmonic valves (absence of diastolic murmur) shows us that the murmur probably depends upon a constriction lying above them; as, if they were so malformed as to obstruct the exit of the blood, they would almost certainly be incompetent also. Now, the certain result of constriction of the pulmonary artery to any extent, or for any considerable period, would inevitably be a gradually increasing congestion and dilatation of the right ventricle—lengthened period of action being equivalent to increase of constriction; and of the existence of this in the present case we have ample proof in the loud systolic murmur audible over the right apex, and in the distinct jugular pulsation, which shows that the regurgitation



through the tricuspid valve has been so persistent and so great that it has destroyed the valvular action of the venous valves at the root of the neck, so as to convert what must at first have been a mere undulatory movement in the venous current into a distinct pulsatory wave. The occurrence of so great a dilatation of the right ventricle sufficiently explains the absence of an apex beat, because the dilated right ventricle, lying in front of the left one, acts as a water cushion or buffer in preventing the apex from reaching the thoracic wall; while the absence of distinct pulsation in the *scrobiculus cordis*, and at the lowest part of the sternum, proves that the heart is feeble, and that in the right side dilatation is the prevalent lesion.

Congenital constriction of the pulmonary artery may occur at various periods of intra-uterine life, and the mechanical results on the cardiac development and consequent symptoms must vary accordingly. When it occurs just previous to the completion of the ventricular septum, and to such an extent as to prevent the complete formation of that septum, we have as a natural result the phenomenon of a complete double circulation with free intercommunication between the two ventricles, one of which is connected with an obstructed, and the other with a patent arterial conduit. The natural result of this is, that when both ventricles contract with nearly equal force upon nearly equal contents, that one whose natural out-flow is obstructed forces a portion of its contents through the abnormally patent channel into the other ventricle whose outlet is unimpeded; and the consequence of this is, forcible dilatation of the unimpeded outlet,—in this case the aorta,—and consequently a systolic murmur of tension accompanying the blood wave, and followed by a loud accentuated aortic second, the natural result of the forcible closure of the aortic semilunar valves by an unusually heavy column of blood, precisely the usual condition in this case. When, however, from any cause—such as catarrhal congestion—the pulmonary circulation is more than usually obstructed, the result is the

same as in the normal condition: the blood-recoil upon the pulmonary valves is greatly increased, and we have an apparent and temporary transference of the accentuation from the aortic to the pulmonic second sound—a phenomenon which repeatedly occurred in this case while the patient remained under observation.

As to the systolic murmur in the mitral area, the absence of distinct propagation round the left side to the back is opposed to the idea of any considerable regurgitation through the mitral valve. The conditions necessary for the production of this regurgitation were, moreover, entirely absent: because free communication between the ventricles prevents the possibility of over-dilatation of the left ventricle and regurgitation from this cause; while congenital mitral constriction, which might give rise to it, would not only be a very unusual complication in such a case, but is also conclusively disproved by the entire absence of any evidence of dilatation of the left auricle, which in all such cases is sure to be revealed by distinct pulsation to the left of the pulmonary area and in the same plane, evincing dilatation and hypertrophy of the left auricular appendix. The systolic murmur over the left apex is therefore in all probability due to propagation from the aortic and pulmonary areas, and possibly also to propagation from the opening in the upper part of the ventricular septum, which I have shown most probably exists.

In this most interesting case, therefore, there exists contraction of the pulmonary artery above the valves, with deficiency of the interventricular septum at its upper part, dilatation of the right ventricle with destruction of the venous valves at the root of the neck, and dilatation of the ascending aorta; and these conditions may be accepted as facts conclusively proved by the phenomena present. But there is one other malformation, the presence of which is rendered probable by what we know of the sequential development of the heart, though it is not, and, so far as I know, cannot be, revealed by any diagnostic phenomena, and that is, more or less imperfect



occlusion of the foramen ovale. Not only, therefore, has it been shown that in this case the symptoms depend upon congenital malformation,—an opinion which even a cursory inspection of the patient would lead almost everyone to adopt,—but it has also been shown that the physical signs present confirm this view, and that these are precisely such as our knowledge of the sequential development of the heart and of the mode in which these physical signs are produced, would lead us to expect to be present in a case of pulmonary constriction arising at a certain period of intra-uterine gestation. The ease with which we can now interpret the symptoms and signs in a case so complicated as the present, compared with the difficulties which formerly beset the diagnosis of the simplest valvular lesion, exhibits in a most striking manner the very great advance in the diagnosis of cardiac disease which has taken place during the last thirty years.

I have only to add that, under appropriate treatment, the patient left the Infirmary improved in every respect, and that she still remains under observation. We see her occasionally, and her condition remains unchanged.

Every case of congenital constriction of the pulmonary artery must vary more or less from every other, though a loud, rough murmur localised in the pulmonary area, dating from childhood and accompanied by cyanosis, may be accepted as pretty conclusive proofs of its existence. While the nature of the coexisting phenomena and the probable lesions upon which they depend may be ascertained, with greater or less certainty, by cross-examining the case somewhat in the fashion I have just described.

In a few very rare cases a pulmonary systolic murmur is followed not by the usual second sound, but by a diastolic murmur. Dr Warburton Begbie has published\* one such case which occurred several years ago in this Infirmary. In this case the lesion was congenital, the pulmonary valve had four segments instead of three, and was incompetent as ascertained

\* Beale's *Archives of Medicine*, No 5.



after death, which resulted from an accident. Such a condition of parts is probably one of the rarest of lesions, but one which there cannot be any difficulty in detecting. I mention it just now mainly for the purpose of warning you against being led into mistaking an auricular diastolic murmur for a pulmonary diastolic one. I have already\* pointed out that mitral stenosis is not infrequently associated with a diastolic murmur, apart and distinct from its own peculiar presystolic murmur. Now and then this diastolic murmur of auricular origin has its position of maximum intensity at the sternal end of the fourth rib, a position in which it might readily be mistaken for a pulmonary diastolic murmur, and possibly has been so mistaken. But in all such cases there is never any systolic pulmonary murmur, such as has been observed in all hitherto recorded cases of pulmonary regurgitation, the murmur in these cases being always double. Moreover, all the ordinary signs and symptoms of mitral stenosis accompany this auricular diastolic murmur, and these, if duly regarded, suffice to keep our diagnosis right. A considerable number of such cases are of rheumatic origin, and the peculiar position of maximum intensity in these seems to be associated with commencing aortic regurgitation which in many of these cases is ultimately fully developed.

I have just one remark to make in conclusion, and that is that systolic pulmonary murmurs, as well as aortic ones, are occasionally produced by pressure from without, from tumours, or more rarely from disorganised lung or intra-pericardial fluid. In these cases we have the absence of all the phenomena already referred to as indicative of other modes by which this murmur may be produced, and we also occasionally can detect the morbid growth or alteration of structure by percussion; now and then, however, the diagnosis is sufficiently obscure, and must be made mainly *per viam exclusionis*, which after all leaves it entirely a matter of probability.

\* Lecture V., p. 134.

## LECTURE IX.

## ON THE VARIATION AND VANISHING OF CARDIAC MURMURS.

GENTLEMEN,—It not infrequently happens that a patient presents himself with a note from his ordinary medical attendant, stating that so-and-so labours under cardiac valvular disease, and yet on careful examination no murmur can be detected. What are we to say to this? Are we wrong, or has the medical attendant been mistaken? Our daily experience in the ward supplies an efficient answer to this most important question; for I think I may safely say that not a single day passes without your having an opportunity of observing the very remarkable manner in which even murmurs dependent upon recognised organic lesions change and vary, and not infrequently disappear, the lesion of course still remaining. This is a most important fact, and one which has been only too much overlooked in the consideration of cardiac disease. It has led, as I have already told you, to many an unlucky *contretemps* not very creditable to practitioners of medicine, nor, let me add, very reassuring to patients who, being one day condemned to a lingering death in life as it were, and the next told by an equally competent practitioner that they are only dyspeptic or hypochondriacal, are apt—and very naturally so—to feel even more sceptical as to medical diagnosis than as to therapeutics. It has led, as I have already mentioned, to the awkward fact of a patient dying of organic cardiac disease, yet possessed of quite a bundle of



certificates from competent medical authorities testifying that he had no cardiac disease whatever.\*

But while ignoring the fact that even the murmurs dependent upon incurable cardiac lesions may vary or even vanish has led to such deplorable results, ignoring the equally important fact that many murmurs, apparently due to incurable lesion, may actually depend upon lesions which are perfectly curable, and in which, therefore, the murmur may not only vanish, but the heart itself become perfectly rehabilitated, has led, in my own experience, to even more lamentable results. It is now many years since a patient of my own, dyspeptic and hypochondriacal, and with a mitral systolic murmur due to simple dilatation of a slight character, consulted a physician, who led her to understand that she laboured under an incurable disease of the heart. Foolishly connecting this diagnosis with sudden death as the inevitable result of any exertion, she from that moment laid herself upon her sofa, and the greatest persuasion could never again induce her to take any exercise other than a rare and occasional drive in a Bath chair. In no long time her dyspepsia was cured, her health restored, her heart rehabilitated, and her murmur gone; but still her watchful avoidance of all exertion remained the same. As may be naturally supposed, she became ere long extremely obese, and after twenty years of positive inaction died of apoplexy, having thrown away her life from a foolish faith in a rash diagnosis. In the course of these lectures I have so repeatedly pointed out the variability and vanishing of murmurs, that it seems at first sight scarcely worth while to say more on the subject; but, indeed, it is a subject upon which too much cannot be said, and, moreover, it is of importance not only to point out that murmurs are variable, but also to show how we may make our diagnosis without unduly depending upon them.

You are to remember, then, that murmurs are by no means dangerous in proportion to their loudness—often the reverse;

\* Lecture IV., p. 120.



and that they may disappear under two distinct circumstances : first, because the heart becomes rehabilitated, sufficiently restored to enable it to carry on its work not only without revealing any sign or symptom of a lesion, but without there being any lesion to reveal ; and secondly, the murmur may disappear, the lesion remaining. Complete rehabilitation is a matter with which only the tricuspid valve is at all popularly credited among physicians ; yet there is no valve which may not be subject to it, though, of course, it is only certain conditions of the valve that are capable of undergoing this change. In the lectures upon "Curable Mitral Regurgitation,"\* and upon "Curable and Incurable Tricuspid Regurgitation,"† I have already pointed out the various conditions under which rehabilitation of these valves may occur. I need, therefore, say nothing further of these at present, except to reiterate the statement, that complete restoration to health after the perfect development of regurgitation—through either, or even through both valves—is a much more common thing than most medical men are at all aware of. The permanence of this restoration is, however, a widely different matter, and depends very much upon the age of the patient, his habits, and the character of his occupation, as well as upon the nature of the original cause of the dilatation ; so that, though the prognosis of the future condition of such a patient is in many cases most hopeful, it is not always so. In aortic regurgitation the condition of the parts is much less favourable for rehabilitation ; but it sometimes happens, though the restoration is much less permanent, however complete it may be for the time. And in speaking of rehabilitation of the aortic valve, I do not refer to those curious cases which occasionally occur,—in which, apparently from deposition of fibrine upon the diseased valves, the regurgitant murmur gradually diminishes in intensity, and sometimes entirely ceases to be audible,‡ the

\* Lecture VI., p. 165.

† Lecture VII., p. 190.

‡ Of this Dr Gairdner has recorded one case in the *British Medical Journal* for March 30, 1872, p. 334, and Dr Walshe another, *Diseases of the Heart*, 3d ed., p. 386.

systolic murmur still continuing, though it may vary in pitch and loudness,—but to cases in which, without any coexisting disease of the valve, its segments have been unable to meet from over dilatation of the aortic orifice, the result of dilatation of the aorta itself. In these cases it has been my lot more than once to observe apparently complete rehabilitation of the aortic valve, accompanied by great and manifest improvement of all the symptoms, occurring under the use of iodide of potassium, a remedy which seems to possess the power of promoting contraction of the arterial tissues. In these cases, of course, there is, from the dilatation of the aorta, always well-marked accentuation of the second sound, terminated in the original state of matters by a soft diastolic blowing sound, which occasionally disappears entirely under the influence of treatment.

Murmurs of regurgitation, we know, may occur at any of the cardiac orifices from dilatation of the parts with which they are connected, apart altogether from disease of the valves themselves, and it is under these circumstances that rehabilitation of the valves may take place, accompanied by a perfect restoration of the normal sound, though that—be it first or second—may remain for a time at least somewhat accentuated. This is especially the case with the second sound, and we cannot expect that ever to be removed. It is less frequently the case with the first sound, though accentuation of it is not infrequently an early sign of commencing dilatation of the ventricle; because if the ventricle recovers its tone at all, it invariably does so more perfectly than the aorta,—being, as we can well believe, much less diseased,—and the restoration to health is much more complete in the one case than in the other. But murmurs of obstruction stand in quite a different category. They invariably result from some direct and material obstacle to the onward current of the blood; and though the obstruction may be a cause of cardiac dilatation it is never its result. The consequence of this is, that though murmurs of obstruction may vary very considerably, or may even completely vanish, the lesion still remains, and the cardiac disease proceeds



at an unaltered pace in its downward career. It is of the utmost importance to remember those facts in connection with the prognosis we may have to give a patient, or rather to his friends. In the one class of cases the disappearance of a murmur may be the signal of the re-establishment of health, in the other it is merely an indication of some unimportant alteration in the physical relation of the parts, which either interferes with the development of the murmur, or at all events hinders its propagation to the surface of the chest. It is not the first time that the disappearance of a murmur, under the latter circumstances, has been hailed with undeserved delight as an indication of renewed life; nay, even a treatment has been instituted—I refer to the starvation and depleting system of treating cardiac disease—which could have no other end than a simple masking of the disease by influencing the physical causes engaged in producing the murmur, but which could not possibly affect, except injuriously, the lesion upon which the murmur depended, and undisguised disappointment has been expressed at the coming back of the murmur on the return of a more natural condition of body, though nothing else ought to have been expected.

In my last lecture I pointed out that a systolic murmur in the pulmonary artery, when it depended upon an abnormal alteration in the anatomical relation of the parts implicated, was apt to disappear, temporarily or permanently, whenever the normal relation was restored temporarily or permanently, or whenever, from any cause, the physical conditions were so altered that a murmur could be no longer produced.\* When a pulmonary systolic murmur, however, depends upon actual physical obstruction, variations in the murmur are less likely to occur, and I have never observed any. And I may make the same statement in regard to a systolic aortic murmur dependent upon the same cause—actual physical obstruction; and as this murmur depends either upon physical obstruction or aortic dilatation, which is equally irremediable, to its full

\* Lecture VIII., p. 208.



extent at least, I perfectly agree with Professor Sanders when he says that the variations in intensity are not by any means so frequent in this form of murmur as in those depending upon other forms of valvular disease.\* These variations do occur but rarely, and depend chiefly upon variations in the ventricular force.

But it is in regard to the murmur connected with mitral stenosis that we find the variation greatest in amount and in variety. In the course of my lectures upon this lesion I have entered pretty fully into this matter;† and in the wards you have daily opportunity of verifying these statements, and of observing that mitral stenosis is at least as frequently associated with an entire absence of murmur, with an irregularly intermittent systolic mitral murmur, or with a more permanent mitral regurgitant murmur, than with a presystolic murmur, with which it is popularly more immediately connected. The following is one of the most interesting cases of extreme variability in the murmur depending on the mitral stenosis which has ever occurred to me:—

CASE XXIV.—J. Munro, aged 21, admitted to bed No. 3 of Ward V. on December 14, 1870. This patient was under my care three years previously. At that time he had a well-marked presystolic murmur, but so variable in character that it repeatedly disappeared, even while being listened to. Under treatment he improved greatly in health; and after his discharge, he was repeatedly seen with a slightly hypertrophied heart, and an occasional presystolic murmur. He afterwards fell off greatly in health, and was readmitted with an enfeebled heart, very irritable, in an almost constant state of irregular palpitation, and with a permanent systolic murmur. He was again discharged somewhat improved, but with the systolic murmur still persisting. At the above date he was readmitted on account of catarrhal symptoms, aggravated by privation and

\* "On the Variations and Varieties of Organic Valvular Murmur."—*Ed. Med. Jour.*, January 1869, p. 584.

† Lectures IV. and V., pp. 110, 134.

want of shelter, and after treatment his murmur was found to have entirely disappeared. On the 1st February 1871 he was exhibited at the Medico-Chirurgical Society without a trace of murmur of any kind. There was some cardiac hypertrophy, a prolonged first sound, and an accentuated pulmonary second. The case was more particularly interesting and instructive, inasmuch as, from changes which took place in the distribution of the wards, he was under the care of Dr Grainger Stewart for some time; and Dr Stewart was able not only to testify as to the persistence of the murmur of mitral regurgitation at the time of his discharge, but also as to the complete disappearance of the murmur at the time of his exhibition to the Medico-Chirurgical Society.\* You see, then, that cardiac murmurs are liable to very great variations, and sometimes vanish entirely beyond the ken of very much more accomplished auscultation than that of that hypothetical individual the average medical practitioner, the lesion still persisting. And you can readily understand how important it is to know how we may determine whether we can in any given case—such as the hypothetical one I commenced with—ascertain the existence of a cardiac valvular lesion in the absence of any murmur; and this, fortunately, is not so very difficult. If you have paid attention to my remarks you will understand that aortic systolic murmurs may vary somewhat in intensity, but never disappear entirely; that aortic diastolic murmurs never disappear even under treatment, unless in the very rarest instances, and in these they are always associated with a systolic murmur, which persists and reveals the lesion; that pulmonary systolic murmurs, if organic, are persistent, if non-organic, they may disappear temporarily during deep inspiration, but only vanish permanently with their cause; that tricuspid regurgitant murmurs are most frequently temporary, and when they vanish, it may be accepted as an indication that their cause has ceased, and the heart has become rehabilitated. Tricuspid obstructive

\* *Vide* "Ed. Med. Jour.," March 1871, p. 832. July 1880.—This patient still lives with his cardiac condition unchanged.



murmurs are rare; and any that I have seen have neither varied nor vanished. So that the greatest interest in regard to the variability and vanishing of organic murmurs centres in the mitral valve, and that, fortunately, is a valve, the condition of which we have the most ample and varied means of ascertaining, apart from the existence of any murmur. And it is fortunate that it is so, for so much discomfort sometimes centres round even its slighter ailments, that not very long ago I had occasion to see a gentleman, thus obscurely affected, who travelled all the way from South America for no other reason but to obtain a definite opinion as to his exact organic condition, which was subject of dispute among his medical advisers in that far off region.

The most distinct and most permanent indication of organic lesion of the mitral valve is an accentuated pulmonary second sound; and in the absence of any other evident cardiac lesion, or of any congestive pulmonary affection, persistent accentuation of the pulmonary second, accurately determined, may be accepted as a certain indication of a mitral lesion. In one case\* this accentuation was the sole detectable indication of cardiac disease, and it was many weeks before we could obtain confirmative information by detecting a mitral murmur, although we sought for it daily. Yet this is comparatively rarely the case; more usually there are other indications, which we only require to feel along the lines certainly to detect. As an indication of how this is to be done, let me give you a case which is not altogether hypothetical. Suppose you have a patient whose lower extremities are somewhat œdematous,† both radial pulses equal, regular or possibly irregular, and distinct, though not jerking, even when the wrist is elevated. These facts teach us the possibility of the heart being affected,

\* Case of Elizabeth Baillie, aged 19, admitted to Ward XIII., March 29th, 1873; discharged May 31st, 1873.

† I here narrate a tolerably well-marked case, but the same method of diagnosis applies to all, the degree of certainty attained varying with the distinctness of the facts.



auricle which is present. Aortic regurgitation has its own distinguishing pulse, and also produces decided consecutive changes on the left ventricle, which are wanting in this case. Aortic obstruction is incompetent to produce such an alteration of the auricle as we have in this case; and the changes it produces, if extreme, involve the left ventricle even more than the right, which is not the case here. *Per viam exclusionis*, we therefore arrive at the mitral valve as the only possible position of an obstruction efficient to produce the phenomena observed. This obstruction to the circulation may either consist in a permanently dilated, or in a permanently constricted, auriculo-ventricular opening. In the former case regurgitation is the sole cause of the obstruction, in the latter, regurgitation, though always present in some degree, plays a very small part in preventing the onward flow of the blood, this being mainly due to the constriction of the opening. With a dilated opening there is no positive obstruction to the onward flow of the blood, which is only hindered constructively by so much escaping backwards which ought to go forwards. As regards the circulation, the results of this lesion are similar to those of aortic regurgitation, while they are somewhat different as regards the heart itself. In aortic regurgitation, as I have already pointed out, the primary result of the regurgitation is the dilatation of that cavity—the left ventricle—into which it occurs; the secondary result is the hypertrophy of the ventricle, without which the circulation would speedily come to a standstill through asystole, the product of over-dilatation. Should the dilatation be great enough to open up the mitral valve the heart is relieved, the chances of death from asystole lessened, the evil day postponed, and, as a rule, death finally brought about by secondary causes. In mitral regurgitation the ventricular changes are of a different character, and are seldom so great. It may be dilated from the tendency of the blood to accumulate in its cavity, and it is often hypertrophied from the increased force required to maintain this circulation in the face of a permanent, though only constructive, obstruction, but

these changes are never great unless the right side of the heart is also largely implicated, so that the heart has an entirely different shape from that found in aortic disease where the alterations are chiefly confined to the left ventricle. The changes in the auricle more immediately concern us. The regurgitation backwards from the ventricle, occurring at the moment when the auricle is commencing to fill from the lungs, tends to dilate that cavity; but this tendency to auricular dilatation is partly counteracted by the patency of the pulmonary veins, which prevents the concentration of the regurgitant force on the auricle alone, and permits its diffusion throughout the whole vascular area of the lungs. Congestion of these organs, of the right side of the heart, and of the general venous circulation, are well-known results of mitral regurgitation; but great dilatation of the left auricle is a comparatively rare phenomenon in such cases. And so also is auricular hypertrophy. There is no actual obstacle to the passage of the blood into the ventricle, which, on the contrary, is even more easy than natural, from the permanent patency of the auriculo-ventricular opening. No increased expenditure of force is therefore necessary to force the blood onwards from the auricle; the slight auricular hypertrophy which may be present is not therefore produced by this cause, but may be regarded rather as a mere counteraction of the tendency to dilate of necessity, forced upon the auricle by the regurgitation from the ventricle. It is otherwise, however, in the case of stenosis of the auriculo-ventricular opening. Here we have not only a continual regurgitation through the permanently patent orifice in the valve which maintains the auricle and lungs in a state of constant repletion, but we have superadded to this a positive obstacle to the onward flow of the blood from the diminished size of the ventricular inlet. From these circumstances there is naturally a greater degree of congestion of the auriculo-pulmonary circuit, and a much greater tendency to dilatation of the auricle, the part naturally most susceptible of this alteration; and from the necessity of forcing the



blood through a more or less constricted orifice, the auricle is always more or less hypertrophied. It is, therefore, as the result of constriction of the auriculo-ventricular opening that we have the auricle attaining its greatest dimensions both as to dilatation and as to hypertrophy, and these are rarely noteworthy at all except in these circumstances. There is, therefore, little room for doubt, that the considerable dilatation and hypertrophy of the left auricle in the case under consideration is due to an actual disease of the mitral valve producing constriction of the left auriculo-ventricular opening. Further, as this great alteration in the left auricle, with the very considerable change in the right ventricle, and in the circulation generally, have taken but a short time to be produced,—only five years having elapsed from the patient's first attack of rheumatism,—the probability is that the mitral opening is of no great size, and the prognosis in the case is certainly grave. You perceive, then, that, without putting the stethoscope to the chest at all, we have been able to make a distinct and apparently most probable diagnosis, to deduce an evidently accurate prognosis, and that we are from the same data able to propound a rational treatment. Could the stethoscope enable us to do more? Certainly not. Would the stethoscope alone enable us to do so much? Certainly not so readily as we suppose. Had we to trust to the stethoscope alone in such a case, what would it teach us? We know that the murmur supposed to be distinctive of mitral regurgitation—of dilatation of the left auriculo-ventricular opening—is a systolic murmur heard loudest over the cardiac apex. And we also know, that the murmur supposed to be distinctive of mitral obstruction is a murmur termed presystolic, and actually occupying the latter part of the diastole, a murmur running up to the apex beat. But this so-called presystolic murmur is, as we have already shown, that murmur most frequently absent even when the physical cause exists of which it is distinctive. Further, there are many cases in which mitral constriction exists along with a murmur which is not presystolic, but systolic in its rhythm,



or even without any murmur at all. If we trust, then, to auscultation alone, as it is generally understood and applied to the heart, that is, if we attempt to diagnosticate the exact nature of any given cardiac lesion by the discovery and discrimination of murmurs, assigning to each its appropriate physical cause in accordance with the position on the cardiac area at which it is best heard, as well as with its rhythm or relation in time to the several acts which constitute a cardiac pulsation, without being actually misled, we shall yet often fail in attaining an accuracy of diagnosis, which is perfectly possible and frequently important. And there is no kind of case in which this is more likely to occur than in that now under consideration. We see, then, that in the case before us, as well as in many others, the stethoscope alone may fail us, and be unable to give us such distinct and positive information as shall enable us to form an accurate diagnosis, and yet we are enabled to do this by a due attention to facts, which were equally accessible to our forefathers in the prestethoscopic ages. These facts owe their diagnostic significance to the diverse modes in which each individual cardiac lesion is compensated; they vary, naturally, in degree, according to the gravity of the existing lesion; their nature is always the same for similar lesions, presenting in this respect a most marked and important contrast to the acoustic phenomena too commonly implicitly relied upon, as these vary not only in degree, but are also often loud and well marked where no important lesion exists, and even more frequently absent when serious lesions are present.

A careful attention to what we may term the physio-pathology of the heart is, therefore, of the greatest importance in the discrimination of its diseases.

Morbid anatomy reveals a diseased structure; pathology unravels the process by which that structure became diseased. What I have termed physio-pathology, or pathological physiology, reveals the consecutive changes which occur, not as part of a morbid process, but are secondarily induced to compensate

the results of that process. Thus rheumatism may shrivel the aortic valves, but it does not hypertrophy the left ventricle. That is a purely physiological change induced by a pathological cause. It affords, therefore, an apt illustration of what I mean by the term physio-pathology of the heart, of which it and the other relative changes constitute one phase.

While, therefore, fully acknowledging the great value of the stethoscope in the diagnosis of cardiac disease, I beg you to remember that it is not all-important, that in order fully to understand its revelations we require also to pay careful attention to the manner in which those consecutive alterations occur, which never fail to follow each special cardiac lesion ; and that, in many cases, a thorough knowledge of these alterations will enable us to form a correct diagnosis apart altogether from any acoustic phenomena which may or may not be present. And that by feeling along the lines indicated, by our knowledge of physio-pathology, we shall always be enabled to give a rational account of our case, both in regard to diagnosis and prognosis, though it may not always be so simply and easily unravelled as the one I have just related.

## LECTURE X.

ON INTERMITTENCY AND IRREGULARITY OF THE PULSE, AND ON  
PALPITATION, CARDIAC AND AORTIC.

GENTLEMEN,—There is perhaps no single symptom connected with the heart which gives rise to so much discomfort, uneasiness, and feeling of insecurity, as intermittency or irregularity of the pulse; yet both of these phenomena are perhaps at least as frequently associated with the absence of serious disease of the heart as the reverse, and this is especially the case with intermittent action. Nevertheless, intermittent action of the heart is so alarming in itself, and so frequently occurs in those who are otherwise of a highly nervous temperament, that it not infrequently constitutes a very serious complication, almost worthy of being regarded as in itself a disease.

Simple intermittence is the slightest form of derangement of the cardiac action, and consists in the occasional omission of a pulsation, the next recurring at the usual period, without any alteration of the cardiac rhythm. The intermission may occur once every two beats, or once every twenty, forty, or more; it may consist in the omission of only one pulsation, or two or three pulsations may be omitted each time, or one pulsation may be generally omitted, and at occasional times we may have the intermission extending over two or three pulsations, thus introducing the element of irregularity in its simplest form. In these cases we have the pulsations equal



however, nervous ganglia are formed in the substance of the heart, and to the influence supplied by these ganglia most modern physiologists seem inclined to attribute the ordinary rhythmical and apparently spontaneous movements of the heart. It is true that if we divide a frog's heart longitudinally, each half goes on beating as usual, while if we divide the ventricles transversely, it is only the upper part containing the ganglia which continues to beat, the lower part remains motionless. At first sight this experiment seems to warrant the conclusion that the ganglia are the source of the rhythmic movements of the heart; but we hesitate to accept this conclusion when we find that the embryo heart of a chick beats rhythmically long before any ganglia are formed, and that the adult heart of a snail goes on beating all its life without any ganglia at all. And our doubts are not lessened when we find that the motionless part of the heart, which contains no ganglia, can still be incited to rhythmic movements, indistinguishable from normal pulsations by the application of an appropriate stimulus. This stimulus may be either an unusual excitement such as the constant current; or it may be a nutritional agent even less perfect in its nature than normal blood, it may be serum or dilute blood.\* These experiments seem to show that the diffuse *vis insita* of the embryo is still active in the adult heart, and is quite capable of maintaining the normal rhythmic movements under normal conditions. And they also show that the ganglia—nervous centres—which are found in the *sinus venosus*, the septum and walls of the auricles, and in the auriculo-ventricular groove, are not only formed to co-ordinate the heart with the rest of the organism through the agency of the cerebro-spinal and sympathetic

of nerve-cells or ganglia, and the results of their experiments on such hearts lead them to regard the cardiac ganglia not as places where nervous energy is produced, but as really *co-ordinators*.—*Proc. Roy. Soc.* vol. xxiii., No. 160, p. 313; and *Ed. Med. Jour.* Oct. 1875, p. 370. This is a view which I entirely homologate, *vide Introduction to the Study of Medicine*, Edinburgh 1865, p. 97.

\* Mennowicz, *Ludwig's Arbeiten*, 1875, p. 132, and *Foster's Physiology*, third edition, p. 170.

nervous systems, but that they also form storehouses of surplus energy, which may be called upon when nutrition is temporarily defective or unusual exertion is required. Of the former condition a state of syncope supplies a common and well-marked example, while less common instances are to be found in the experiments of Panum and Von Bezold already referred to\* and in the beating of the heart of a shark or frog hours after its severance from the organism to which it belonged; while instances of the latter condition are to be found in those cases where the heart is incidentally called upon for unusual exertion, as happens in many forms of disease.† But besides acting as storehouses of surplus energy, the cardiac ganglia also co-ordinate the cardiac movements in accordance with the multifarious requirements of the organic frame in its ever varying states of health and of disease. Without entering at large into the anatomy and physiology of the nerves of the heart, it may suffice to say that an accelerating agency seems to be conveyed to the ganglia from the sympathetic system, mainly through the cervical and first dorsal ganglion, and this excito-motor influence not only accelerates the cardiac pulsations, but, according to Von Bezold, also increases their force. The *vagus*, on the other hand, by means of its inferior cardiac branch, inhibits and slows the heart's action, and when excited arrests the heart in diastole. While the superior cardiac branch of the *vagus* is regarded as really an independent nerve passing upwards to the medulla oblongata in the same bundle with the *vagus* fibres, and conveying from the heart a controlling influence to the nerve centres there which regulate the movements of the arterioles. This nerve is supposed to come into action where the heart is oppressed with work by a rise in the arterial blood tension, it inhibits the regulating centres, dilatation of the arterioles follows, the blood pressure is lowered, and the heart relieved. Experimental physiology leaves no doubt as to the substantial cor-

\* *Vide* page 81, note.

† *Vide* page 84.



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\* Memnowicz, *Ludwig's Arbeiten*, 1875, p. 132, and *Foster's Physiology*, third edition, p. 170.



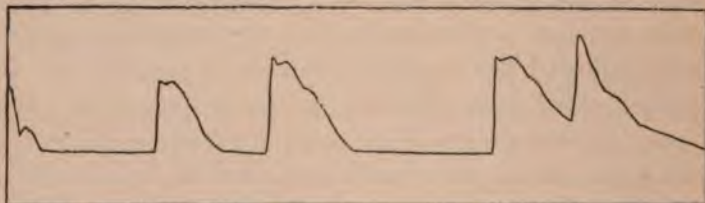
nervous systems, but that they also form storehouses of surplus energy, which may be called upon when nutrition is temporarily defective or unusual exertion is required. Of the former condition a state of syncope supplies a common and well-marked example, while less common instances are to be found in the experiments of Panum and Von Bezold already referred to\* and in the beating of the heart of a shark or frog hours after its severance from the organism to which it belonged; while instances of the latter condition are to be found in those cases where the heart is incidentally called upon for unusual exertion, as happens in many forms of disease.† But besides acting as storehouses of surplus energy, the cardiac ganglia also co-ordinate the cardiac movements in accordance with the multifarious requirements of the organic frame in its ever varying states of health and of disease. Without entering at large into the anatomy and physiology of the nerves of the heart, it may suffice to say that an accelerating agency seems to be conveyed to the ganglia from the sympathetic system, mainly through the cervical and first dorsal ganglion, and this excito-motor influence not only accelerates the cardiac pulsations, but, according to Von Bezold, also increases their force. The *vagus*, on the other hand, by means of its inferior cardiac branch, inhibits and slows the heart's action, and when excited arrests the heart in diastole. While the superior cardiac branch of the *vagus* is regarded as really an independent nerve passing upwards to the medulla oblongata in the same bundle with the *vagus* fibres, and conveying from the heart a controlling influence to the nerve centres there which regulate the movements of the arterioles. This nerve is supposed to come into action where the heart is oppressed with work by a rise in the arterial blood tension, it inhibits the regulating centres, dilatation of the arterioles follows, the blood pressure is lowered, and the heart relieved. Experimental physiology leaves no doubt as to the substantial cor-

\* *Vide* page 81, note.

† *Vide* page 84.

rectness of this estimate of the functions of these cardiac nerves, and it has also taught us that all the vagaries of an allorhythmic heart are most probably to be explained by an interference of the opposed action of the inhibiting and accelerating nerves, though no doubt it may be a little difficult to explain why in any given case, at any given time, one form of irregularity should be present rather than another. At another time I hope to enter on this subject with more minuteness; at present I shall only treat it in the most general and practical way. It may be well, however, in passing, to point out that the exact nature of the irregularity is not always to be discovered by reference to the radial pulse alone, and perhaps I could not do this in a more striking manner than by showing you the three following tracings. The first of these, you will see, is a very well-marked example of a

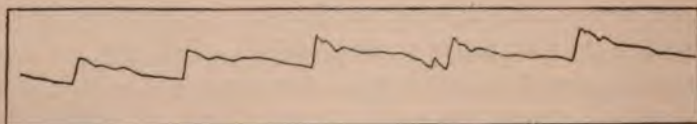
Fig. 17.



bigeminal pulse, taken from the radial artery of a patient suffering from aortic regurgitation not long before his death.

In the second tracing, taken also from the radial artery, there

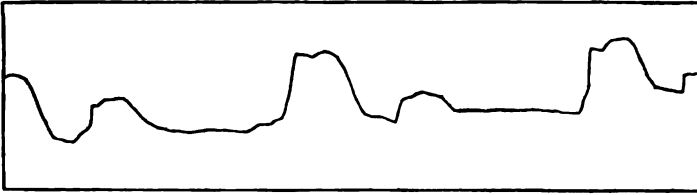
Fig. 18.



is no appearance of a bigeminal rhythm at all, yet this is at once revealed by the accompanying cardiogram which was taken simultaneously. In the latter case the twin beat of the left ventricle was too feeble to reach the periphery, hence it is absent in the radial tracing, yet this case was the most inter-

esting of the two, being a well-marked example not only of bigeminal pulsation but also of that still more rare form of irregularity in which the right and left heart predominates alternately, in which, therefore, as you see, we may have a

Fig. 19.



radial pulse presenting nothing very unusual, and yet the carotid artery and jugular vein may be seen to pulsate alternately.\*

It is somewhat remarkable that, so long as the heart remains mainly under the influence of the *vis insita*, and unaffected by extraneous agencies acting through its nervous connections, that is, so long as the foetus remains in utero, intermittent action is unknown, it must occasionally occur, because, even in utero, agencies are now and then at work which ought to produce it. But its occurrence must be very exceptional, as I know of no such case recorded, and have inquired at several friends, much in the habit of auscultating foetal hearts, without ascertaining that intermittent action has ever been observed in such cases. But no sooner is the child born than injurious agencies multiply and increase in intensity, and by their reflex action it comes to pass that we have intermittent cardiac action developed in so many instances as to constitute infantile intermittence a well-known fact. And from man's birth to his grave it is a phenomenon of frequent occurrence, though in itself it has as little influence in precipitating his exit from this world as in influencing his entrance on its stage. The late Dr Mathew Baillie was once consulted by a patient with an extremely irregular pulse, and being pressed to give a reason

\* Case of Euphemia Lawson, referred to at page 185.



for its occurrence, he replied, "I don't know what is its cause, but I can tell you this, it won't shorten your days." The patient, a native of this city, was forced to content himself with this opinion, and lived to prove its truth, for he died at a very advanced age many years subsequently. But it is not always so innocuous; for, while it generally indicates weakness of the cardiac walls, and often some irritability of the cardiac nerves not always of much importance, so it many times is also associated with serious lesion of the valves, or of the cardiac muscle, which does tend to shorten life.

I have already mentioned\* to you that extremely irregular action is almost pathognomonic of mitral stenosis; Mr Adams of Dublin† regarded it as quite pathognomonic of this lesion, provided the irregularity were of such a character that several of the faint fluttering cardiac pulsations every now and then failed to be felt at the wrist. In this he was no doubt wrong, as irregularity of this character is at least as common in the gouty heart, whether dilated or not, as in mitral stenosis; and he was also wrong in following Corvisart and attributing extreme irregularity in both force and frequency of pulse to narrowing of the aortic orifice. Elliotson corrects this opinion, for he says, "I do not happen to recollect a pulse irregular in force and frequency in a single case of narrowing of the aortic opening solely, while I know that it is very common in the narrowing of the left auriculo-ventricular opening, though possibly not peculiar to it, nor indeed to narrowing of any opening."‡ My own experience leads me to coincide with Elliotson's opinion, which I believe will be found to be correct. To constitute it pathognomonic of mitral stenosis, irregular cardiac action need not present any special form of irregularity, for that varies in each case, but it must be associated with some, at least, of the other concomitant phenomena diagnostic

\* Lecture V., p. 136.

† *Dublin Hospital Reports*, vol. iv. p. 494.

‡ *On Various Diseases of the Heart*, fol. London, 1830, pp. 16, 17.

of this lesion, and most certainly with accentuation of the pulmonary second sound, a phenomenon not always easily recognised in such cases, because both second sounds are apt to be much weakened when the cardiac action is extremely irregular, still in mitral stenosis it will be always possible to discriminate the pulmonary second as a shade more distinct than the aortic second, though it may not be markedly accentuated. Very frequently in such cases the existence of an irregular mitral systolic murmur simplifies the diagnosis, though this is by no means always the case. In all such cases there is also evidence of cardiac failure from pyrexia or overwork, frequently a history of rheumatism, and very often well-marked signs of pulmonary and of general venous congestion. I know of no cardiac cases more amenable to proper treatment than these are, none in which relief is so certain of attainment, though of course perfect cure is unattainable, and even complete removal of the irregularity is hardly to be expected, though it is sometimes temporarily obtained. In all such cases the pulse is of course permanently small and feeble even when it is not irregular, and this—as well as the presence of pulmonary accentuation—constitutes an important point in the diagnosis between irregularity associated with mitral stenosis, and that depending simply upon gout. In the latter class of cases, with which we may associate atheroma of the coronary arteries, also a frequent cause of cardiac irregularity we have of course no accentuation of the pulmonary second; in the intervals between the fits of irregularity the pulse is of the normal fulness and strength, and this is also the case with those pulsations which during the attack fairly reach the radial artery. In mitral stenosis, then, while some of the cardiac pulsations fail entirely to reach the radial artery, even those which do reach it are small and feeble; but in the gouty heart, while some of the pulsations also fail to reach the radial artery, most of those which do reach the periphery are of average strength and fulness. The chief exception to this rule is when the gouty heart is already dilated and the mitral valve incom-



petent, but in these cases we have, superadded to the irregular action, all the ordinary signs of dilated hypertrophy to guide our opinion. Once fairly developed there is probably no class of cases, not associated with absolute organic disease, in which a permanent cure is so rarely attained as in gouty irregularity, though they are susceptible of great relief from appropriate treatment, and with moderate care are not usually attended by any considerable shortening of the normal span of life; some, indeed, with even extremely irregular gouty hearts, continue in fair health to a very advanced age without any obvious impairment either of bodily or mental vigour, except that depending upon senescence.

There is one form of abnormality, however, in a gouty heart, which I do not hold to be so susceptible of a favourable prognosis. I refer to that to which attention is ordinarily first directed by the occurrence of symptoms referrible to cerebral anæmia, in which abnormal slowness of the pulse is the most remarkable symptom—a pulse which is slow, not because the cardiac action is retarded, but because many of the pulsations fail to reach the periphery. It is long since Hope pointed out that, though cardiac action was occasionally really slow, yet the bulk of those cases in which the pulse was said to range from 20 to 30 a minute, were really cases in which one or two of the ventricular beats were regularly and permanently imperceptible in the pulse, every second or third pulsation only reaching the periphery;\* and Dr Stokes has made some most important and instructive remarks on the same subject, expressly in connection with the production of cerebral anæmia, and all the peculiar and important phenomena which may result from that.† In one very instructive case which I saw some years ago, an old lady, long gouty, though without regular attacks, and with a dilated feeble heart, had been suddenly seized while shopping with what seemed to be an epileptic attack. These attacks continued to recur when she made the slightest exer-

\* *On Diseases of the Heart*, London, 1839, p. 337.

† *The Diseases of the Heart and the Aorta*, Dublin, 1854, p. 362, &c.



tion, and at the time I saw her she was unable to rise from the recumbent posture without bringing on one. They were associated with flatulent dyspepsia \* and an extremely slow pulse, averaging about 20 per minute. On careful examination I found that her heart acted with perfect regularity, but with unequal force, so that the apparently abnormal slowness of the pulse was simply due to the fact that only about every third beat reached the periphery. It was not difficult to connect this defective cardiac action with a diminution of arterial pressure sufficient to produce all the phenomena of cerebral anæmia on the one hand, and among these epileptiform seizures, and also defective secretion generally, and as one of the results of that flatulent dyspepsia. There is but one remedy that I know of capable of remedying this state of matters, if indeed the muscular fibre be still perfect enough in structure to permit its being remedied, and that is digitalis, full doses of that were prescribed, and with nutritious diet and a fair allowance of stimulants, it succeeded so well in her case that within a week she was going about as usual, was so well, indeed, as to give a dinner party, and she died gradually from asthenia some years subsequently, without any return of these serious symptoms. Such cases are, however, always more serious than those of simple irregularity of the cardiac action and are not always so easily remedied, considerable degeneration of the cardiac muscle being only too frequently concomitant.

Irregularity of the pulse is, you will see, most frequently associated with mitral stenosis or with gout; no doubt it is also associated with other cardiac affections, or with apparently simple dyspepsia, rarely however, I think, unless these affections or that dyspepsia be associated with the gouty diathesis. Simple intermittence, however, stands in a somewhat different category, sometimes it is only an early indication of failure of cardiac power dependent upon anæmia, over-work or worry, or upon valvular disease or gout, but it is often a

\* *Vide* Lecture XI.

purely nervous phenomenon. In the former class of cases we have the affection commencing by an intermission followed by a thump; as the disease progresses this thump becomes associated with a sensation of tumbling, and by-and-by the irregularity and inequality of the heart's action reveals itself to the sufferer by a rapid and irregular succession of thumps and tumbles of varying force. In the class of cases, however, to which I now refer, the disease never progresses beyond the thumping stage, and the thumps are not even very distinct; the patient has at the most an uncomfortable sensation in his cardiac region of varying intensity, lasting for less than a second, and if we happen to feel the pulse at that moment we become aware that this uncomfortable sensation is associated with the omission of a radial pulsation and nothing more, and sometimes this takes place regularly without the patient being in any respect conscious of it. Such cases are of purely nervous origin, often arise from sudden fright, grief, or anxiety, and the intermission thus produced, though for a time of frequent recurrence, gradually dies out, and sometimes disappears entirely, while in some it remains permanent, though much more infrequent than at first. This form of intermittence originates in the accidental coincidence of fright or anxiety with a heart congenitally or acquiredly feeble, or with a nervous system from similar causes unduly impressionable. I have known it commence with the shock of a railway accident, and in that case the intermissions were at first every second beat, but in a few months came down to one intermission in twenty beats, and I have no doubt will ultimately disappear. Richardson has known it follow shipwreck in one instance, with a somewhat similar result, and sudden grief, anxiety, or anger in several other cases.

But emotional inhibition of the heart is often a much more serious matter, and may fatally arrest its action; this probably never occurs in those whose hearts are structurally sound, but it has happened often enough in those who were not known to labour under cardiac disease to make it a possible occurrence



to any one past middle life, and to some even at an earlier period. Zimmermann \* has published a number of such cases, and from him we learn, what we would otherwise scarcely have expected, that unexpected joy is more dangerous to life than sudden grief. Sophocles died suddenly of joy on being crowned, at the age of ninety, as the first tragic poet of the age. The same thing happened to Phillpides the comic writer. Chilon of Lacedæmon died in the arms of his son, who had borne away the prize at the Olympic games. The famous Fouquet died on being set at liberty by Louis XIV. The niece of Leibnitz died suddenly of joy at finding a box containing ninety thousand ducats below the philosopher's bed. And many similar cases are recorded by Zimmermann and others, all testifying to the fatal effects of excessive emotion, even when of a pleasing character. It seems more natural to expect that terror and grief should be more injurious than joy, and though this does not seem to be the case, yet these emotions have in their turn been fatal to many. Philip II., king of Spain, enjoys the unenviable notoriety of having frightened two of his counsellors to death. One of his ministers of state died suddenly on being sharply rebuked for a hesitating answer. Another, the Cardinal Espinoza, died a few days after being sternly told, "Cardinal, know that I am master!" Mallet's tale of Edwin and Emma was founded upon an actual incident that happened at Bowes in Yorkshire, in 1714, when a young woman died suddenly of grief on being told of the death of her lover. Palmer, a celebrated comedian of last century, died on the stage of the Liverpool Theatre on August 2, 1798, while performing the part of the "Stranger" in the play of that name. Palmer had recently lost his wife and a favourite son, and when one of the *dramatis personæ*, in the course of the play inquired for his wife and family, Palmer, unable to reply, became inexpressibly agitated, and dropped dead. In the end of last century Prince George Louis of Holstein, having removed the body of his wife from

\* *A Treatise on Experience in Physic*, London 1782, vol. ii. p. 268, &c.



one coffin to another of more costly materials, desired his valet to read him some pages from a pious book, and, kneeling by the side of the coffin, he burst into tears and died. And a couple of years ago there occurred in France an even more startling instance of the fatal effect of overwhelming emotion. Dr Deleau, a celebrated aurist, only forty-four years of age, leaning over his dying daughter to receive her last farewell, himself fell dead as if struck by lightning. The effects of emotional inhibition vary with the violence of the emotion on the one hand, and the sensitiveness of the organism on the other ; in some individuals the result dies rapidly off with the fading of the emotion which produced it, while in others the disturbed rhythm of the heart persists as a fact of which the sufferer cannot for one instant lose the consciousness, and which ultimately wears him out by the silent but terrible and sleepless suffering which it induces. Yet this emotional inhibition, so varied and often so terrible in its results, is simply an exaggeration of what we have all probably felt at one time or other when our heart has, for the moment, stood still in the face of any impending danger to ourselves or others. It is merely an aggravated form of what our vernacular poet Ballantyne has so graphically described as indicative of maternal anxiety:—

“ My vera heart gaes loup, loup,  
Fifty times a-day.”

This “loup” being nothing but the perceptible thump—the forcible systole—which succeeds a momentary intermission. In highly impressionable individuals, now and then, there is no returning thump, the intermission becomes permanent, and we have sudden death from emotional inhibition of the heart. Emotion, when it thus acts, is reflected from the sensorium to the cardio-inhibitory centre in the medulla oblongata, and from it to the heart, apparently through the inferior branch of the vagus. When a sensory nerve is implicated in the inhibition of the heart, consciousness does not seem to be necessary for its development, though it is probable that it materially increases the

severity and danger of the inhibition, hence the great importance of relieving pain if it be severe, especially in weakly and impressionable individuals. Severe cold, if applied to the surface suddenly, especially in those exhausted by fatigue, is no infrequent cause of fatal inhibition of the heart. During the Russian campaign (1812-13) the French soldiers were often seen to fall dead as if struck by lightning from the effects of the excessive cold. At Smolensko more than thirty grenadiers of the Italian Guard fell dead from this cause, in attempting to set themselves in line on the height beyond the Borysthenes.\* A cold bath has often proved fatal to infants in this way, and many a one has died in the water from an inhibited heart. These unfortunates are said to be seized with cramp and to drown; they really sink dead in the water, the heart fatally inhibited by cold; now and then, however, such victims do struggle, and are partly drowned, for inhibitory paralysis of the heart, like every other form of asystole, is not necessarily instantaneously complete, but may be ingravescent in its character. Cold, applied internally, is equally well known as a cause of fatal cardiac inhibition. The knowledge of this fact is of very ancient date indeed, for Quintus Curtius tells us that Alexander the Great lost more of his soldiers from their drinking the ice-cold water of the Oxus, than he did in any one of his many serious battles; and almost every summer some death in the harvest-field, from a similar cause, supplies a fresh modern illustration of the sudden way in which life may be brought to a close by a shock thus applied. One hundred years ago the pathology of such cases was very much based upon Tristram Shandy's idea that the body and soul are like a coat and its lining; if you rumple the one, you rumple the other also. The violence of an emotion constituted its danger. "A passion," says Zimmermann, "without even being carried to excess, will sometimes occasion a difficulty of breathing,

\* *A Treatise on the Effects and Properties of Cold*, by Moricheau Beaupré, M.D. Translated by John Clendinning, A.B., M.D. Edinburgh: Maclachlan and Stewart, 1826, p. 149.



together with a sense of stricture in the breast, and an hesitation to speak, the tongue remaining as it were immovably fixed to the palate. The weaker passions speak, the stronger passions are mute."\* Greater violence of emotion was supposed to collapse the heart, so that it neither received nor emitted any blood, the sufferer dying instantaneously;† and it was also known, as in the case of Philip V. of Spain,‡ that the heart occasionally ruptured from similar causes. Essentially popular in its character, this pathology was of very ancient date, and, when Shakespeare makes Malcolm say to Macduff—

"Give sorrow words ; the grief that does not speak  
Whispers the o'erfraught heart and bids it break,"

he but expresses tersely in poetic form the weak periphrasis of Zimmermann. Since the beginning of this century men have ceased to be contented with vague descriptions of still vaguer ideas, but have sought to connect physical results with definite physical causes. Among the earliest pioneers in this direction, in regard to the influence of injuries of the nervous system on the heart, were Legallois§ and Wilson Philip.|| Yet, though the pathological acumen of Alison¶ distinctly recognised the bearing of the experiments on the cardiac depression, so often observed in the early stages of abdominal inflammation, as well as their relation to sudden death from a blow on the epigastrium, we had got no further forty years ago than to connect fatal cardiac failure with concussion of some part of the cerebro-spinal or sympathetic system. It is then entirely within the last forty years, and chiefly within the last twelve, that we have learned to recognise with any precision the mode

\* Op. cit. p. 267.

† Loc. cit.

‡ Zimmermann, op. cit. p. 274.

§ *Philosophical Transactions*, 1815 and 1817. *Vide An Experimental Inquiry into the Laws of the Vital Functions*, London 1818, 2d edition.

|| *Outlines of Pathology, and Practice of Medicine*, 1844, Edin. p. 12.

¶ *Experience sur la Principe de la vie, notamment sur celui des mouvement du Cœur et sur le siège de ce Principe*. Paper read before the Institute of France, June 3, 1811. *Vide Œuvres de le Gallois*, Paris 1830.



in which the heart's action is influenced by extraneous impressions. And though we owe this largely to foreign observers, of whom we may especially mention the Brothers Weber,\* Cyon,† and Von Bezold,‡ yet the experiments of Professor Rutherford§ have done much to clear away obscurities, and to give us distinct ideas as to the innervation of the heart, and particularly as to the inhibitory influence of the vagus nerve. Much still remains to be done, but we have certainly gained a firm basis from which to extend our inquiries, and on which to base our treatment.

In the treatment of intermittent or irregular pulse, we must be guided very much by the condition of the patient, and the existence of any actual cardiac disease, or of any irritation capable of reflexly producing such irregularities, and this we must carefully ascertain for ourselves, and never trust to the mere statements of the patient. Because there is nothing more common than for a dyspeptic patient to say, "I never have a headache, I may eat and drink what I please, my stomach never troubles me;" true, but his heart does, and careful examination will discover that his stomach is not so perfect as he represents it to be. It is precisely the same with him as with a patient with neuralgia of the shoulder joint and a decayed molar tooth; "You need not look there," he says, "I never have toothache," but he winces when we touch the tooth, and if we get leave to extract it his neuralgia is cured. The one man has toothache in his shoulder, the other dyspepsia in his heart; the cases are analogous, and teach us to put more faith in our own careful examination than in the statements of any patient, which, let me add, however, there is no need to

\* *Omodei Annali Universali di Medicina*, vol. exi. pp. 225, 233.

† *Bericht d. Sächs. Ges. d. Wiss.* 1866, s. 308.

‡ *Untersuchungen über die innervation des Herzens*, Leipzig 1863. *Vide also Untersuchungen aus dem physiologischen Laboratorium in Würzburg*, Leipzig, 1867.

§ Paper read before the Royal Society of Edinburgh, May 1869, published in abstract in the *Journ. of Anat. and Phys.* vol. iii. 1869, p. 402, and in full in the *Trans. Roy. Soc. of Ed.*, vol. xxvi., 1872, p. 107.

contradict; we are bound to cure our patient if we can, but it would be both thankless and dangerous to attempt to confute all his prejudices.

In the intermittent pulse of infancy and childhood little treatment is required; the bowels must be regulated if necessary, but more by food and exercise than by medicine, for whatever enfeebles the frame tends to keep up the intermittence. We must also by moderate exercise in the open air, early hours, plenty of sleep, and the use of a nutritious but unstimulating diet, seek to tone down any nervous instability, and to develop a state of rude unconscious health. The patient ought to be warmly clad, and the use of quite cold water as a bath avoided, as any nervous shock ought to be most carefully shunned. If other remedies seem needful the bromide of iron is a very useful one, or in very irritable patients it may be necessary to have recourse to the bromide of ammonium, for a time at least.

In patients affected with cardiac disease, we of course regard the irregular pulse as a mere symptom, and treat the central lesion upon which it depends, whatever that may be. In by far the larger proportion of cases it will be found to be mitral stenosis, as I have already told you, and as the irregular action accompanying this lesion is merely a sign of cardiac debility, what we require to do in these cases is simply to steady the heart's action, increasing at the same time the force of its muscular contractions. All this we can do most effectually by the judicious use of digitalis, so much so that often in a few days the patient will express himself as feeling a new man, and he will not much mind any little remains of irregularity, which it may be difficult, if not impossible altogether to remove. Of course though digitalis in small, repeated, tonic doses, must be our main stand-by in these cases, other drugs as subsidiary agents are frequently of great importance, and must be used *pro re nata*; such as carbonate of ammonia when bronchitic rhonchi are present; squill if there be much cedema, if only a little we may safely trust its removal to the digitalis; arsenic



if there be much cardiac pain, which there seldom is in these cases; bromide of ammonium, with or without morphia—subcutaneously or otherwise—if there be much nervous restlessness; and iron in some form or other if anæmia be a prevalent symptom.

When, however, intermittent or irregular cardiac action comes to be a subject for medical treatment apart from cardiac disease, the heart may still be soothed and steadied by small doses of digitalis if necessary, but the treatment falls mainly under three heads: *first*, to remove the cause if possible; *second*, to brace up the whole organism; and *third*, to soothe the nervous system. If the cause be mental, such as grief or anxiety, all our cure will frequently be baffled, and our success will usually depend not so much upon our remedies as upon our influence, and also upon the amount of mental firmness originally possessed by our patient, and whether he is still capable of being roused to exertion. In nervous shocks from anger or fright we have a potent help in the narcotic needle, which, timeously employed, abbreviates the period of shock and lessens its subsequent influence, besides giving the patient confidence in our resources and in their power to relieve him; the dose of morphia injected must, however, be a full one, and such as is sufficient to ensure sound sleep for some hours. We must in every case caution the patient against all depressing agencies, such as excess in venereal pleasures, in tobacco smoking, in work, especially intellectual work; we must avoid all worry or excitement of any kind, and we must prescribe abundance of sleep, fresh country air, plenty of sunlight, perfect quiet, light amusing occupation, and nutritious diet in small quantities at regular intervals, suited to the requirements and capacities of our patient. But no solid food should be given at a less interval than four or five hours, so as to avoid introducing fresh food into a stomach still containing undigested material, as nothing is more injurious; but a tumblerful of hot water, or a large teacupful of hot solution of Liebig's extract of beef, washes out the stomach, prevents the accumu-



lation of flatulence, and often proves most useful in stimulating the completion of digestion and the emptying of the stomach. This may be given about an hour or so after a meal, or when symptoms of uneasiness come on, and a fresh secretion of gastric juice is effectually secured by alkalising the draught with a small teaspoonful of carbonate of soda.

Alcohol is the one domestic remedy which exercises the most potent influence upon an irregular and intermittent heart;\* it is, however, one which must be used with caution, because excess in its use is apt to perpetuate and increase the very evil it is employed to cure. Still, moderately employed, its action is not only palliative, but to a certain extent curative; only it must be employed in moderate doses, and in those forms which contain fewest substances likely to disagree, and these are in the main good sound claret, and pure whisky free from fusel oil or all injurious impurity. Next to these comes sound sherry, neither too dry nor the reverse, but of medium quality. Porter, ale, and beer are useful enough at times, as well as all other alcoholic fluids, but as a rule are not to be commended, though in every case we must be guided by the idiosyncrasy, the purse, and the convenience of our patient. The object we seek to attain is to provide a nutritive, diffusible stimulant, slightly narcotic or sedative in its qualities, and one the components of which shall disturb digestion as little as possible, while we also take care that the quantity introduced shall not be sufficient materially to interfere in this way. About two ounces of absolute alcohol is the most that can be introduced into the system in one day without detriment, but this may be given in divided doses, and in various forms according to the requirements of the patient. Coffee, but especially tea, are excessively injurious to such neurotic patients, they ought therefore to be avoided. Cocoa is too heavy, but cocoatina often agrees well, though, as a rule, a French breakfast, with meat, fruit, and claret, will be found to

\* In this I may seem to follow Dr Richardson, but the fact was well known to me before the publication of any of his papers.

suit such a patient much better than any other; while hot soup with a little well boiled rice will answer equally well in most cases. For lunch a tumblerful of milk and Carrara water, a glass of beer, or a basin of soup, according to taste. For dinner plain roast or chop, a single mealy potato, or spinach, avoiding other vegetables, no pudding, and two or three glasses of claret, or a couple of glasses of sherry, will be found very serviceable. No tea, and at night a glass of whisky and potass water, with or without a biscuit. This is a sort of model diet for such cases, which must be varied to suit each individual case. We must, especially in this climate, secure that whatever is taken in the morning be sufficiently stimulating to enable the patient to withstand the cold—especially in the winter—and to soothe him under the unavoidable worries of life. To this end, if soup be preferred for breakfast, a glass of sherry, or a tablespoonful of brandy stirred into the white of an egg previously dissolved in a little water with a pinch of sugar, will be found a most agreeable and useful lunch. What we seek to attain by the use of alcoholic stimulants is the temporary soothing of the vagus nerve, the temporary rousing of the heart to more vigorous action by which it is enabled to resist evil influences, the improvement of the digestion, and the gradual accumulation of vital energy. What we must most carefully avoid is any excess in the use of alcohol, which would most surely lead to the production of a catarrhal condition of stomach, or to its keeping up, if it already exists, as to that more perhaps, than to anything else, is due the persistence of intermittent cardiac action. But in these cases alcohol is really a remedial agent, from which we can obtain more good than from any other drug, only it must be used as a drug with caution. Definite rules only apply to definite cases, but there are three grand rules which apply to every case, and these are, that the alcohol must be given in a digestible as well as stimulating form, in divided doses throughout the day, and never in excess, otherwise we shall increase the evil we desire to cure.



What we desire to do in such cases is to brace up the general system, at the same time protecting it from injurious influences. Warm clothing, therefore, is a necessity, and cold sponging of the chest every morning; but bathing, particularly sea-bathing, must be shunned as dangerous, the shock being only too apt to produce, in such cases, fatal inhibition of the heart,—cramp as it is so often called,—which is so instantaneously fatal that the patient sinks, but is not drowned; he is dead before he sinks.

The drugs which will be found most useful vary with each case, pepsine in ten or fifteen grains with each meal seems to do good always, but beyond that we must be guided by subsidiary symptoms. If there be much catarrhal irritation of the stomach, nitric or nitro-hydrochloric acid and taraxacum; calumba, or quassia are often useful, as this condition is often kept up by a congested condition of the liver which the acids tend to relieve by promoting the bile secretion. It is always useful in such cases thoroughly to alkalisise the stomach at least once a-day by the free use of soda, potass, or lime in the form of lime water. With this treatment we may combine the use of podophylline in quarter grain doses with a third of a grain of ipecacuan and a quarter of a grain of belladonna, which in enfeebled patients unloads the liver, and relieves the right side of the heart without purging. If torpor of the liver be more marked, then small doses of blue pill and aloes, just sufficient gently to move the bowels, are most useful. If torpor of the colon be the chief apparent ailment, the long-continued use of Barbadoes aloes in small doses with sulphate of iron, hyoscyamus, and nux-vomica, answers very well, and if much flatulence be present we may substitute a couple of grains of the compound galbanum pill for the hyoscyamus with advantage; or if gout be the fundamental ailment, then small doses of the acetic extract of colchicum with Barbadoes aloes, both in such doses as shall ensure no more than one stool, a little more bulky than usual, and continued daily or every second day for some time, will be found most useful.



When a hæmatinic tonic is required, as will be the case in most instances, the citrate of quinine and iron will be found to be the mildest, and the one most generally useful, while Easton's syrup of the phosphates of strychnine, quinine, and iron, is the most powerful, and if continued in drachm doses twice a day for several months, will often effect a most wonderful improvement in the patient's health, and in the state of his heart; that it may do so we must be careful to have all catarrh of the stomach removed in the first place, and the liver also acting freely, otherwise this tonic will not only do no good, but occasionally seems to do harm. It may frequently be combined with two minim doses of the liquor arsenicalis with much advantage.

Whenever, from the state of the patient and the defective secretion of urea, gout seems to be impending, the most important remedy will be found to be the free administration of colchicum along with alkalies, and in those cases plenty of Vichy or Apollinaris water will be found most useful, accompanied by a rigid abstinence from animal food, an abstinence, however, which must be guided by reason and not by prejudice, as many patients have a difficulty in nourishing themselves when altogether deprived of an animalised diet.

Although for temporary purposes there is no sedative equal to the subcutaneous injection of Squires' solution of the bimeconate of morphia, yet for continuous use as a nervine sedative, bromide of ammonium far surpasses it, but it must be given in full doses, from half a drachm to a drachm three times a-day, till its full sedative effect is secured. This is but a bare outline of the treatment to be adopted in those cases, but from it you will see, that for the relief of intermittent and irregular cardiac action, we must endeavour first to determine the lesion upon which they depend, cardiac or otherwise, and we must treat this with due regard to the organic debility to which that lesion owes its injurious efficiency, and we must meanwhile not forget that between the cause and its effect we have the nervous system as a connect-

ing link, and that, by modifying or interrupting this connection, which we often can do by the judicious use of sedatives and narcotics of various kinds, we may cause to cease, or at all events mitigate, the results pending our attempts at cure.

Cardiac palpitation is only too frequently dependent upon similar causes, as irregular action, and is to be treated accordingly, especially by such means as shall restore a normal tone to the heart and to the organism generally. Now and then an apparently accidental though violent attack of palpitation seems dependent upon acidity of the stomach, and can often be at once relieved by an antacid draught of soda, potass, or ammonia; and indeed not only palpitation, but also some of the minor forms of irregular action, are promptly relieved by a draught containing a drachm of aromatic spirits of ammonia with or without an equal quantity of tincture of valerian, or failing that, by a tablespoonful of whisky or brandy with a teaspoonful of carbonate of soda in about a wine-glassful of water, just enough not wholly to drown the miller, as we say in Scotland. I need scarcely add that for such patients tea, coffee, and tobacco, especially the latter, are absolute poisons, and must be wholly eschewed for a time at least.

Epigastric pulsation, depending on irritability of the abdominal aorta, is a local neurosis not always apparently dependent on dyspepsia, nor to be relieved by tonics. I have, however, found it almost invariably to yield to full doses of the bromide of potassium in some bitter infusion such as calumba, gentian, or chiretta. The only exception to this that I remember seeing was that of a woman, a patient in Ward XIII., in whom this excessive abdominal pulsation was accompanied by preternatural hardness of that part of the artery, probably due to atheromatous disease, and in her case large doses of the iodide of potassium gave great relief, though nothing had any permanently curative effect.

In connection with the subject of increased cardiac action generally, I may mention that, while increased action is liable to follow any unusual exertion, such as climbing a stair or



going up a hill, both in hearts valvularly diseased, and also in those which are simply weak, palpitation or irregular action occurring while the patient is at rest is by no means to be regarded as a certain symptom that a heart is only weak or gouty, because of course hearts valvularly diseased are always weak, and often gouty, and therefore liable to present the symptoms both of diseased and also of simply feeble hearts. There is, however, one peculiarity by which the valvularly diseased heart may be perfectly discriminated from a simply weak heart, and that is, that while palpitation or cardiac discomfort, occurring as the result of exertion in a heart valvularly diseased, can never be relieved by anything but rest; the same results following exertion in the feeble heart of a nervous or gouty individual are frequently calmed down by any emotional excitement, especially of a pleasurable kind, such as meeting a friend, or the sight of anything novel or attractive, or even, strange to say, by a more violent exertion. Thus a man with a heart merely valvularly diseased is not likely to have any discomfort unless he meet with a slight ascent in his walk, when he is at once brought up and must rest, but a man with a gouty or feeble heart, though he too may be "afraid of that which is high," and may also suffer during the ascent, yet has his palpitation at once relieved by any emotional excitement, and if he be seized with sudden palpitation while walking slowly on the level, he will often find it disappear at once if he takes a short race to the next lamp-post; the heart beats the faster for the exertion, but the palpitation is gone, affording a very peculiar form of inhibition, which probably only those can truly appreciate who have experienced it. This peculiar mode of obtaining relief in such cases seems allied to the tendency of some epileptics to run forwards. I have most often observed this peculiarity in those who, without being epileptic themselves, were yet related to those thus afflicted. I have also to add that there are some sufferers from aortic regurgitation who, even when pulled up by severe angina, can yet by a violent effort throw it completely



off. I have recently seen a very remarkable instance of this.\*

The explanation seems to be that some slight inhibition has temporarily enfeebled the heart's action, the heart is pained from want of proper nourishment, by a voluntary effort a sudden call is made upon its reserve force, and the copious flushing of its tissue with fresh blood reinvigorates the heart, and relieves the pain. I am not aware that relief is ever similarly obtained in the cardiac discomfort of mitral disease.

\* This patient, I may mention, has by appropriate treatment been entirely relieved of his angina.

## LECTURE XI.

## ON THE SECONDARY RESULTS OF CARDIAC DISEASE.

GENTLEMEN,—There are various subsidiary affections which group themselves naturally—from physiological reasons—round cardiac lesions, and now and then some one or other of these affections asserts itself in so marked and determinate a manner as to challenge attention as if it were a primary and independent disease. It is of importance, however, for the patient, sometimes for his safety, and always for his comfort, that we should recognise these ailments as secondary, and by at once attacking the cause, give speedily that relief which, if obtained at all, can only be obtained in a round-about and unsatisfactory manner by treating as a disease what is essentially a symptom.

Some of these ailments are neurotic in character and are not so likely to be misinterpreted; to these I shall by and by more particularly direct your attention. But the larger proportion of the affections to which I refer are due to those alterations in the circulation, which originate with the very commencement of cardiac disease, and upon the more or less rapid development of which its termination depends. These may be placed under two heads: *first*, the diminution of arterial pressure; and *second*, those alterations in the venous circulation which precede the fatal transference of the intra-vascular pressure from the arteries to the veins. As already mentioned, the absence of capillary resistance and of vascular motor nerves within the

lungs make mere mechanical influences play a much more important part in the pulmonary circulation than they do in that of the system generally. Hence any obstruction to the onward flow at once makes itself perceptible by inducing an accentuation of the pulmonary sound,\* and ere long produces dilatation more or less marked of all the pulmonary vessels, with intense congestion and occasionally splenisation of the pulmonary tissue. In such circumstances, as you can readily understand, the blood moves but slowly through the lungs, its mass therefore is imperfectly arterialised, and consequently, with a perfect ability to breathe deeply and freely, the patient yet labours under a most distressing breathlessness. Many circumstances may arise during the course of any cardiac disease which may tend to intensify this breathlessness: such as a greatly increased size of the heart, from dilatation and hypertrophy; effusion into the pericardium or pleura; and what is even more common than either of those conditions, a catarrhal state of the bronchial mucous membrane, or an oedematous state (from serous effusion) of the air-cells themselves, the two latter conditions being specially due to the capillary congestion present in such cases. Of course in all cardiac cases, from the remora in the venous circulation, and its diminished intensity everywhere, there is a tendency to spontaneous coagulation in the dilated bulgings of all the systemic veins, sometimes even in the heart itself, especially in the appendices of the auricles. From these thromboses we frequently have emboli detached which now and then are large enough to plug the pulmonary artery and cause sudden death, but which in the larger proportion of cases merely produce infarctions, which in the lungs give rise to hæmoptysis, limited dulness with localised crepitation, occasionally to pain in the chest, and now and then to a rapid rise of temperature which may rapidly disappear. At other times they give rise to actual attacks of pneumonia in the lung tissue surrounding them. Sometimes, instead of being removed by

\* *Vide* Lecture I. p. 30.



gradual absorption of the blood, as they occasionally are, or instead of shrivelling into a strongly pigmented mass of cicatricial tissue as is generally the case, they die, and we may have all the symptoms of necrosis of part of the lung, with a foul smelling ichorous expectoration, or still more rarely the necrosis and consequent perforation may extend into the pleural cavity, and pneumothorax may result. Now and then we may have hæmorrhage from the lung arising from rupture of a vessel simply from the intense congestion, but much more commonly it depends, as I have told you, upon an embolic infarction.

You see then that, even in the most acute pneumonic attacks, we must never forget the possible cardiac origin of all the symptoms. For hæmoptysis we shall find a cardiac cause almost as frequently as any other. While in cases of frothy serous expectoration, with rhonchi and crepitation in the lungs, we have to look to cardiac debility—if not to some better defined and more serious lesion of the heart—as only too frequent a cause of that state of pulmonary congestion which has intensified the result of some trifling exposure to cold; while cardiac breathlessness presents symptoms so altogether peculiar that they can scarcely fail to be recognised, and the central cause detected.\*

But it is in the systemic circulation that we find the most multifarious phenomena dependent upon the one central lesion, phenomena which are as varied as the functions of every organ of the body, lesion of any one of which is their proximate cause. Confining our attention for the present to the results of defective aortic pressure, I need hardly point out the importance of the pseudo-apoplectic or epileptiform seizures which I have already described† as indicative of failure of the heart's force in various circumstances, or of giddiness, threatened or actual syncope, tinnitus aurium, flashings of light across the field of vision, and occasional flushings of a usually pale face, which the patient describes as "puffs of heat,"

\* *Vide* Lecture I. p. 2.

† Lecture X. p. 258.

as signs of cardiac debility of common occurrence, particularly in aortic regurgitation. And yet these symptoms, coupled as they usually are with an apparently full and bounding pulse, have been referred to cerebral congestion, and treated by leeches or even by venesection to the manifest weakening of that organ, the debility of which was the sole cause of the symptoms described. At the present time it is unnecessary to do more than to point out the cause upon which these symptoms most frequently depend, to indicate the necessity of a careful examination of the heart in such cases, and to refer to what I have already said in regard to the treatment of such cases,\* because it is only by rectifying the central lesion so far as that is possible, that we can expect to remedy the peripheral phenomena dependent upon it.

But there are many other phenomena originating in defective aortic pressure which are less obviously dependent upon it, and which are frequently connected with cardiac diseases less easily detected than aortic regurgitation. Perhaps the most important of all these subsidiary phenomena is diminution of the water of the urine, and its importance is due to its being one of the earliest of these secondary affections to make itself known; unfortunately it is too often known only to the patient himself, and neither to his friends nor to his medical adviser, but there is all the more need to inquire into it. We know very well that one of the earliest results of diminution of the aortic pressure is the diminished action to all those glands whose secretive action is mainly dependent upon the maintenance of efficient arterial pressure, and of these the kidneys are perhaps the chief, they are certainly the earliest to manifest plainly a diminution of their secretion. The salivary glands are perhaps the only other glands the defective secretion of which would be as readily detected, but they, as is well known, are, so far as their secretion is concerned, so entirely separated from the general circulation and placed under local influences, as to deprive their defective secretion of

\* Lecture III. p. 102.

all somatic importance, so far at least as the heart is concerned. But whenever we find the urine persistently diminished in quantity, and its specific gravity unaltered or even increased, and when we find as the result of that a deposit of lithates, apart from any coldness of the weather or any somatic catarrhal or febrile condition, then the state of the central organ of the circulation ought to be inquired into, and it will be certainly found to be defective.

The next most important condition is a flatulent stomach with or without more or less of tympanitic distension of the abdomen. It is astonishing how often this condition is unavailingly treated by tonics, blue pills, antispasmodics, &c. ; the central lesion—even if it be recognised—being altogether ignored as the efficient cause of this subordinate phenomenon. Yet our first inquiry in all such cases ought to be into the state of the urine, as a corroborative indication, and our next and most important investigation ought to be into the state of the heart. It is astonishing how long such subsidiary phenomena as I have described may occasionally persist without progressing further. I have seen a flatulent abdomen and defective secretion of urine last for twenty years without the development of any other or more serious symptom, except very considerable marasmus, which of course was to be expected from the long-continued defective digestion, and I have also seen all the more prominent symptoms in such a case vanish after a few days' appropriate treatment.

If we except the cerebral phenomena, it is comparatively seldom that we find symptoms due to defective arterial pressure apart from those dependent upon venous congestion, and sometimes we find the latter alone predominant. This is specially the case in menorrhagia, which is so frequent a concomitant of heart disease, and which in these cases frequently depends entirely upon venous congestion. We also find, especially in the later stages of cardiac disease, a trifling amount of albumen in the urine, not dependent upon kidney disease, but simply upon serous transudation probably



dependent on a local increase of the intravenous pressure. From the researches of Kürschner\* we know that fluids pass through animal membranes in the following order : *first*, water ; *second*, saline solutions ; and *lastly*, albuminous or gummy fluids ; and Bruecke† has shown that certain membranes allow albuminous fluids to pass more readily than others, and that this probably depends upon the original size of the pores ; while Liebig‡ has confirmed Kürschner's experiments by measuring the force necessary to drive the various fluids through membranes ; but he did more, for he ascertained that, by a continuance of the experiment, the pores became apparently widened, so that after twenty-four or thirty-six hours the force of pressure might be lessened by one-third or even one-half without any diminution of the result. The widening of the pores of any membrane must therefore be regarded as the proximate cause of the passage of albumen through it. This may be produced by force of pressure alone, and from what has been recorded of the effects of intravenous pressure, however produced, this cannot but be regarded as a most efficient source of albuminous transudation ; § moreover, long persistence of pressure is as we see, equivalent to a considerable increase in its amount, and as we learn from Kürschner's experiments that membranes permitting the passage of saline fluids are but one step removed from those permitting the passage of albuminous fluids, so we can readily understand that the membrana propria of the kidneys may easily permit the passage of albumen through it upon any slight increase of the intravenous pressure, such as so constantly occurs in the course of cardiac disease. Only we must remember that diminished secretion is the most sensitive test, that it depends upon defective

\* Wagner's *Handwörterbuch der Physiologie*, Bd. i. s. 62.

† *De Diffusione Humorū per Septa Mortua et Viva*, Berol, 1842, s. 55.

‡ *Untersuchungen über einige Ursachen der Säftebewegung*. Braunschweig, 1848, s. 6. Vide also Henle's *Rationnelle Pathologie*, 1847, Bd. ii. s. 467-8.

§ Robinson *An Inquiry into the Nature and Pathology of Granular Diseases of the Kidney*, London, 1842. Emmert, *Beiträge*, &c., and Henle's *Rationnelle Pathologie*, Bd. ii. s. 458, &c., &c.

arterial pressure, and that to render the increase of the intravenous pressure effective in producing albuminous transudation we must have a certain amount of vascular fulness present. Hence, as I have just said, if there be great marasmus, diminished secretion may persist for many years without the occurrence of albuminous transudation; and, on the other hand, if there be much vascular turgescence, albuminous transudation may be a comparatively early symptom. A small amount of albumen in the urine is therefore an important sign of an increase of the local intravenous pressure, the result of cardiac disease, though if any tube casts be present they must be regarded as probably indicating the existence of independent kidney disease, because we rarely find them occurring in kidneys altogether free from disease,\* while a diminished amount of urine of normal or high specific gravity, with a small amount of albumen, is not found in any independent form of kidney disease of any serious extent. A diminished amount of urine with a large amount of albumen indicates inflammatory disease of the kidneys which has other and more distinctive symptoms; and a varying but usually considerable amount of albumen with a large quantity of nearly normal urine indicates waxy degeneration; while in contracting kidneys, which have a very close and intimate connection with cardiac, and especially with arterial disease, the urine is never more than slightly albuminous, but is always increased in quantity and of a low specific gravity, the urine in these affections differing, as you see, very considerably from that to which I have just referred as indicating mere congestion apart from any actual renal disease.

Again, whenever we have defective digestion from diminished secretion of gastric juice, from a cardiac cause, we have also venous congestion of the liver, diminished secretion of bile, and congestion of the portal circulation, with tendency to hæmorrhoids, diarrhœa, or ascites. The defect of the bile

\* *Der Harn Cylinder und seine diagnostische Bedeutung.* Dr. Burkart, Berlin, 1874, pp. 25 and 82.



intensifies the results of the want of gastric juice, hence we have putrefaction of the ingesta, abdominal flatulence, and constipated bowels, with the passage of foul smelling stools. German authors have asserted that at this stage the bile is reduced in quality by the addition of a quantity of serum (albumincholie), but this is a purely hypothetical assumption, and is not more likely to be true in the earlier stages of cardiac hepatic congestion than in any other case of defective bile secretion from congestion from any other cause; by and by there comes a time, however, when this is the case, but then we also have signs of serous escape elsewhere; in cardiac disease this can only rarely happen first at the liver. As in the deeply congested lungs catarrhal conditions are apt to arise, so also in the deeply congested intestines we are apt to have similar results, but as they are less exposed to the vicissitudes of temperature, they are comparatively seldom affected in the early stages, but occasionally in the later stages of cardiac disease we have a sero-mucous diarrhoea which the patients are prone to term dysentery. The extension, or rather the co-existence, of a similar catarrhal condition of the bile ducts is very apt to produce more or less complete obstruction of them, and so to cause jaundice, a jaundice which from its greenish hue is very readily distinguished as of cardiac origin, the cyanotic hue of the congested skin combining with the bright yellow of the bile pigment to produce this peculiar appearance, and perhaps there are few things more striking than the change from green to bright yellow which takes place on the upper surface of the body after death in such cases from the hypostatic draining of the blood downwards.

Whenever we have long-continued persistent congestion of any organ, there must of course be a persistent turgescence of that organ, increase of size where that is possible, generally also increase in amount of connective tissue present in it. This hyperplasia of the connective tissue is to a variable extent observed in the lungs, much more frequently and to a much greater extent in the liver, to a less extent in the spleen,



and also in the kidneys. Whatever may be the cause of this hyperplasia, it may after a time proceed to contract, and, by destroying the secreting tissue of the organs affected, induce an atrophy of the gland affected. But this is by no means a common result in the congestion of actual cardiac disease, more usually the disease is fatal before contraction sets in. It is true that not infrequently we have contracted kidneys associated with cardiac disease, in such cases it seems most probable that the arteries are primarily at fault (arterio-capillary fibrosis—Gull and Sutton), while the heart is affected secondarily (dilated and hypertrophied from physical causes), and that the kidneys become ultimately affected through the agency of venous stasis. This is certainly the sequence of events in many cases, but the full history of these events belongs to that of arterial disease, which is too wide a subject to be entered on now.

Every cardiac case involves several of these secondary lesions to which I have referred, some less and others more marked than usual. In the following case several of these subsidiary affections will be found conjoined in a very well-marked measure.

CASE XXV.—Elizabeth M'Dermot, aged 40, admitted to Ward XIII., 30th Dec. 1869, complaining of pain in the chest and breathlessness. She stated that since February last she had been subject first to a pain in her right hypochondriac region, and latterly over her heart, and that this had been more severe during the last four weeks. She had never had rheumatism nor scarlatina, but for the last five years she had dysenteric attacks every now and again. She had been to India, but only for a short time, and was always best when at sea. Patient was of ordinary height, anxious expression, lips, gums, and conjunctivæ blanched, yet tinged of a light yellow as was also the waxy skin—the entire absence of the usual greenish hue, depending on the great degree of anæmia present—her pulse was 98, small and feeble. On placing the hand over the cardiac area, a distinct heaving impulse was

communicated to it by the heart's action, and an evident thrill preceded and ran up to the apex beat. The apex beat between the fifth and sixth ribs about three inches to the left of the sternum. The vertical dulness one inch to the left of the sternum commenced at the upper border of the third rib and ran into the liver dulness; the transverse dulness commenced at the right edge of the sternum and extended to the left for four inches and a half. Over the apex a loud, rough, purring murmur was heard preceding and running up to the first sound. In the aortic area both sounds were distinct, the second somewhat feeble; in the pulmonary area the first sound was distinct, the second markedly accentuated. The patient had great difficulty of breathing, complaining especially of oppression on the right side; she had a considerable cough, and expectorated a quantity of frothy serous fluid. The pulmonary percussion was normal; on auscultation, rhonchi and rattles were audible over both sides of the chest. The tongue was pale but tolerably clean, no appetite, considerable thirst; bowels irregular, frequently there was watery diarrhoea, stools pale. The liver dulness extended in the nipple line from the upper border of the fourth rib to about one inch above the umbilicus, a distance of over five inches. The uterus was healthy; the urine diminished in quantity, deposited urates, and contained a trace of albumen. The prognosis from the first was unfavourable,\* chiefly from her anæmic and exhausted condition, and she died exhausted upon the 8th of January 1870, her death having been hastened by a recurrence of the diarrhoea. At the *post mortem* examination the body was not much emaciated, the skin was of a yellow tint. An unusually large quantity of yellow serum was found in the pericardium; the surface of the heart was fatty; the pulmonary valves competent; the segments of the tricuspid valves were slightly

\* As a rule jaundice in cardiac disease is a late phenomenon, and indicates a speedy end. In one case, however, I have known jaundice to last for several years even though concomitant with an ultimately fatal stenosis of the mitral valve.



thickened at their apices; the segments of the mitral valve were thickened, the auriculo-ventricular opening narrowed; the segments of the aortic valve was thickened, but the valve was competent; the aorta was atheromatous; the substance of the heart pale, and apparently fatty. The bronchi were filled with frothy yellow mucus, especially on the left side; both lungs, but especially the right one, had pigmentary deposits scattered through them, their tissue was stained of a yellowish hue, they were otherwise healthy. The liver weighed four pounds five ounces, its tissue was slightly fatty and stained of a yellow colour; the gall bladder was enlarged and full of dark bile, the bile ducts cedematous and blocked with yellowish mucus. On the surface of the right kidney there were many embolic cicatrices, the renal tissue was paler than usual and of a yellowish tint, it was slightly fatty, and the Malpighian corpuscles were slightly prominent. The spleen weighed eight ounces and a half, and had a lobulated surface; on being cut into it was firmer than usual, but there was no alteration beyond simple hypertrophy.

But the general increase of venous turgescence resulting from cardiac disease is not only to be recognised by the alterations in the secretions, and by and by in the structure of the internal organs, it makes itself visible externally in a bluish or purplish tint of the lips, cheeks, and digital extremities, in rarer cases by a dusky tint—cyanosis—of the whole surface of the body, now and then, when the superficial congestion is not extreme, and the skin is delicate, as in young females, a certain amount of oxygenation seems to take place through it in its most exposed parts, and in such cases the cheeks may have all the rosy hue of health, but the digital extremities are always purplish and cold. Indeed, the surface temperature of the body is usually somewhat depressed. Now and then some of the larger external veins become turgid, but the congestion must be considerable before this takes place, and it is not very common in young persons in whom the external veins are usually small. In the neck, as I have already told you, these veins not infrequently



pulsate, and this may even be now and then—though rarely—seen in all the superficial veins. In all cases of cardiac disease the tissues are more or less soaked with serum, while very trifling causes precipitate the occurrence of pulmonary cedema, or pleural effusion. The latter condition is revealed by a rise in pitch in the percussion note, and a diminution in the respiratory murmur; while a slight alteration in the respiratory murmur, without dulness, reveals the existence of pulsary cedema, long before we can hear any crepitation. These changes are more common in mitral than in aortic disease. Sooner or later in most cases anasarca sets in, usually beginning at the most depending part, most commonly at the instep, gradually increasing till accumulated more or less in every cavity of the body, constituting what is called general dropsy. But though the instep is, as it were, the position of election for the commencement of cardiac dropsy, and though it usually commences there, yet now and then when cardiac congestion has lasted for a long time without any very serious cardiac debility, it may happen that cirrhosis of the liver may have been to some extent induced, and then the influence of the cardiac and hepatic obstruction combined are sufficient to cause the dropsy to be ascites, and not anasarca, though the latter is not long of following; this condition is I believe peculiar to mitral disease, and is most common in mitral stenosis.

Embolism is only too common a result of cardiac disease, and gives rise to phenomena of the most diverse character according to the organ affected; paralysis of motion, sometimes temporary, frequently permanent; aphasia, often; hæmaturia followed by temporary albuminuria, not infrequently; pneumonia, or effusion into the pleural cavity, not infrequently; pulmonary infarction and hemoptysis very frequently. Embolism, as you know, is the term applied to a local stoppage of the circulation by the sudden blocking of a vessel by an embolus, that is, any foreign body moving within the vessels; a few air bubbles or oil globules may,

under certain circumstances and in certain places, produce all the effects of more solid masses. More usually, however, embolism is brought about by little bits of fibrine vegetation, broken off from diseased valves, or what is just as common, by bits of clot from thromboses found in the auricles or ventricles of dilated hearts or in the veins. The veins in this case being by no means necessarily inflamed, the dilated bulgings in those veins which are tortuous from long continued congestion being a favourite location for such thrombi, venous remora being, if we may so term it, the active cause of the formation of this blood coagulum or thrombosis, to which a spanæmic condition of the blood, as well as a quiescent state of the patient, may also materially contribute.\* The phenomena of embolism vary in every different case, and depend upon the size of the embolus, as well as upon the organ in which it may happen to be arrested; yet, varied as its results may be, there are probably few pathological processes so simple in their essence, or which can be so readily traced.

Perhaps the most instructive way, not to be tedious, in which I could conclude this lecture, will be to narrate to you the following case, in which there were repeated attacks of embolism of various organs during the patient's residence in the ward, more especially as in itself it is possessed of very considerable interest, and in spite of the absence of a *post mortem* examination, there can be no reasonable doubt as to the embolic cause of her repeated attacks.

CASE XXVI.—J.M., aged 33, a wardress in the Edinburgh Prison, was admitted to Bed 16, Ward XIII., on 17th April 1873, complaining of pain in her left side, extending down her thigh to her left calf, and upwards to her left forearm and hand. These pains had lasted continuously for three months, had been accompanied by repeated diurnal rigors and nocturnal

\* As venous remora is usually a very distinctive mark of gout, there is no wonder that gouty thrombosis and even secondary phlebitis are not uncommon. *Vide* Paget's *Clinical Lectures and Essays*, London, 1875, p. 292, on Gouty Phlebitis.



perspirations, and had been treated as rheumatic by her medical adviser. The patient's family history was good, and she herself had always previously enjoyed good health, except when she suffered from a severe attack of rheumatic fever at the age of fifteen. Her expression was weary and anxious, she was nervous and easily startled, had occasional headaches, and slight hæmorrhages from the nose when it was blown. Her respirations were 32 per minute, pulmonary physical signs normal, no cough nor expectoration. Her pulse was 96, soft, easily compressible; systolic impulse forcible, and falling rapidly off from the finger (Corrigan's pulse)—a phenomenon markedly increased by elevating her arm at a right angle to her body as she lay in bed. The apex beat was full, diffuse and forcible, and pulsated below the sixth rib, two and a half inches from the left edge of the sternum. At the level of the fourth rib dulness commenced one inch to the right of the sternum, and extended across the chest for a distance of five and a half inches. The cardiac vertical dulness did not rise above the third rib, but the aortic dulness extended to the upper edge of the sternum, and the aortic pulsation was distinctly to be felt in the tracheal fossa. On auscultating in the mitral area a rough murmur was heard running up to the first sound which was obscured, but not replaced by a systolic murmur which, when traced upwards, was found to have its position of maximum intensity in the aortic area, where it was loud, rough, and followed by a soft blowing diastolic murmur, which completely replaced the second sound. In the pulmonary area a distinctly accentuated second sound was heard, preceded by a systolic murmur propagated across from the aorta, and somewhat obscured by a diastolic murmur traceable to the same region; a distinct double murmur was audible in both carotids. Her tongue was clean, appetite moderate, bowels regular. The urine was slightly opalescent, and of a pale straw colour, moderately acid, without deposit, but containing a trace of albumen; sp. gr. 1026. The patient had not menstruated for several months.



In this case there could be but little dispute as to the diagnosis; obviously we had a constricted mitral opening from disease of the mitral valve, and an aorta dilated with diseased and incompetent valves, these being not atheromatous to any extent, probably rather shrivelled, both affections being due to her former attack of rheumatism, her present attack being possibly but doubtfully of rheumatic character. Prognosis moderately hopeful as to her present attack; serious, of course, as to the ultimate issue of her cardiac disease.

The double murmur in the carotids indicated great freedom of regurgitation, and therefore absence of any great thickening or abnormality of the aortic valves, and the disease being evidently of rheumatic origin, and unaccompanied by any great dilatation of the aorta itself, it was obvious that these valves must be somewhat shrivelled, to permit of so free a regurgitation as existed. While the double murmur in the aortic area indicated local obstruction both to egress and ingress, and pointed therefore to deformity of the valves accompanied by some degree of stiffening, and possibly with vegetations on their under surface. Had the valves been comparatively normal, so greatly incompetent as to permit of a regurgitation as in this case, the double murmur would have been audible in the arteries alone, with a single diastolic murmur below the level of the valves, and with a comparatively mute aortic area. The persistent localisation of the neuralgic pains was opposed to the idea of their purely rheumatic character, while the alternating rigors and perspirations were more neurotic than rheumatic in their character, and the condition of the urine agreed with this supposition. We subsequently had reason to believe that this attack was probably due to embolism. She continued to improve till the afternoon of 17th May when she had a sudden rigor; she was much distressed, and complained of intense headache. Her temperature rose suddenly to  $105^{\circ}$ ; her pulse 112; her respirations, 40. The urine contained a trace of albumen; chlorides apparently undiminished. On examination of the

chest at the base of the right lung posteriorly, a small localised patch was detected over which fine crepitation was audible at the end of the inspiration. No dulness on percussion; no bronchial breathing; no cough, nor expectoration. Ordered a jacket poultice to the chest, and ten minims of chloroform in one drachm of olive oil every four hours.

18th May.—Temperature, *mane*  $104^{\circ}6$ , *vespere*  $103^{\circ}6$ ; respirations, 40; pulse, 108. Distinct crepitation audible on inspiration over the base of the right lung; no dulness; no bronchial breathing, or bronchophony; slight cough; no expectoration.

19th May.—Temperature, *mane*  $102^{\circ}$ , *vespere*  $101^{\circ}8$ ; respirations, *mane* 36, *vespere* 28; pulse, *mane* 96, *vespere* 88; crepitation on inspiration continues. Urine slightly albuminous; chlorides undiminished; tongue still foul.

20th May.—Temperature,  $98^{\circ}8$ ; respirations, 32; pulse, 84; crepitation has disappeared. Pulmonary physical signs altogether normal.

The suddenness of this attack, and its equally sudden recession correspond to the peaked ephemeral type of pneumonia—a form of pneumonia occurring, as Wunderlich has pointed out, chiefly in connection with trifling local processes, and frequently of embolic origin. In this case the embolic origin of this very sudden and sharp attack was at once recognised and pointed out, no credit being taken for cutting short a disease the pathological course of which is frequently so brief. Yet many such cases have no doubt served to bolster up the idea that severe pneumonia can be cut short by such perturbative treatment as free venesection; had we bled our patient freely, with what chucklings might we not have recorded the obvious recurrence to the good old type in her pneumonia, and its remarkable amenableness to appropriate treatment.

The patient remained well till the evening of 1st June, when she was suddenly seized with sickness and vomiting. Tongue foul; headache intense. Nothing abnormal was found



anywhere except in the urine, which was smoky, with a red deposit of blood corpuscles, as revealed by the microscope; no tube casts were observed this evening, but the examination was somewhat hurried. The urine contained about one-seventh of albumen; chlorides undiminished. A mustard poultice was applied over the epigastrium, and small bits of ice were directed to be swallowed.

2d June.—Patient much in same state, vomiting continues, and headache intense. Urine bloody, sp. gr. 1015, albumen about one-seventh; epithelial and granular casts were to-day observed for the first time, and in considerable number. Pulse, *mane* 92, *vespere* 104; respirations, 37; temperature, *mane* 100°·2, *vespere* 101°·6. *Treatment*:

R Potassii iodidi,  
Ammonii bromidi, aa ʒij.;  
Infusi chimaphilæ, ʒvj. M.

Sign.—Capiat æger ʒss. tertiis horis ex aqua.

3d June.—Headache still severe; vomiting ceased; a vesicular eruption beginning to appear on the forehead and cheeks; urine unchanged. Pulse, *mane* 114, *vespere* 120; respirations ranging from 32 to 36; temperature, *mane* 102°, *vespere* 103°·4. Omit iodide.

4th June.—Eruption well-marked, forming transparent bullæ, ranging in size from a split pea to that of a half marble, and full of limpid fluid. Headache lessened; urine unchanged; tube casts more numerous, and more of them granular; tongue dry; pulse, 108; respirations, 36; temperature, *mane* 105°, *vespere* 104°·2.

5th June.—Headache has disappeared; urine unchanged; patient complains of no pain, but is evidently intensely ill. Pulse, *mane* 116, *vespere* 114; temperature, *mane* 102°, *vespere* 102°·4; respirations, *mane* 41, *vespere* 34.

After this she continued to improve till 13th June, when her pulse was 86, her temperature 99°·4, and her respirations 25. Tongue clean and moist; appetite good; the blood has disappeared from her urine, which only contains a mere trace



of albumen, an occasional granular tube cast being still to be detected. The eruption, which was obviously due to the iodide of potassium, has now almost disappeared. This sudden attack of hæmaturia, unaccompanied by any of the local signs of renal congestion, was obviously due to the arrest of an embolus in the kidney, and the subsequent local hæmorrhagic infarction which accompanied it. After the failure of the iodide of potassium the treatment was purely expectant, and consisted chiefly in the free administration of diluents.

On the 16th of June her temperature was  $98^{\circ}4$ ; respirations, 24; pulse, 92. At the evening visit on that day, while the patient herself was under examination, her right hand and arm were observed to twitch convulsively, and the patient, who had just been interpreting the language of an aphasic neighbour, became suddenly sick, and complained of violent headache, chiefly on the right side.

Next day (29th June) she felt better, but still complained of headache, and could not lift her head from the pillow without becoming sick. Temperature,  $99^{\circ}$ ; pulse, 100; urine free from blood, and containing only a trace of albumen; sp. gr. 1020. Nothing abnormal was observed.

21st June.—Pulse, 100; temperature,  $100^{\circ}8$ ; tongue very foul; urine darker than usual, with a copious deposit of phosphates, and almost free from albumen. The abducens muscle of the left eye is to-day observed to be sluggish in its movements, so that a slight squint is occasioned.

The patient continued to improve till 28th June, when the squint had almost entirely disappeared, the abducens muscle having almost completely regained its normal power. This sudden attack of local paralysis could obviously be ascribed to nothing else than a small embolus arrested in the brain, which broke up quickly, and was removed without producing anything more serious than a temporary paralysis of a single muscle; the convulsive movements of the right arm were, like the headache, probably due to reflex irritation.

On 3d July, at 11.20 A.M., this patient was suddenly seized

with violent headache, chiefly referred to the occipital region, and this was followed by severe vomiting ; her eyes were fixed at first, but this passed off, and the patient retained her consciousness for a time, till, after a violent fit of retching, sudden unconsciousness set in, followed by gasping inspirations, which gradually became less and less frequent, the face becoming more and more livid, the patient dying at 1.30 P.M. During this final attack the right carotid was observed to beat with very much greater force than the left one. It was very obvious that here we had first a small cerebral embolism followed by a much larger one on the right side, which cut off so much blood from the brain as to produce immediate unconsciousness and speedy death from asphyxia, the respiration being interfered with by abnormal innervation ; there was no syncope, the heart beat forcibly to the last. No examination of the body could be procured ; but the symptoms of each attack were so plainly of an embolic character, while her cardiac lesion supplied so sufficient an explanation of these emboli, that the pathological history of this most interesting case could scarcely have been made plainer by a dissection.

## LECTURE XII.

UPON PAROXYSMAL ANGINA PECTORIS AND OTHER FORMS OF  
CARDIAC PAIN WITH SOME REMARKS ON THE DIAGNOSIS OF  
FATTY HEART.

GENTLEMEN,—Pain in the cardiac region is not only a common symptom in all diseases of the heart, but is often enough complained of when no cardiac disease is present; it is also an important indication of substernal aneurism. Pain below the left mamma is well known to be an almost constant complaint in chlorotic and debilitated—anaemic—individuals, whether male or female; and many neurotic individuals, whose nervous system is developed at the expense of their muscular, who are often said to be gouty, and frequently are so, complain of various uneasy sensations in the cardiac region, often amounting to a positive soreness of the heart. In some cases the infra-mammary pain is wholly external, a form of intercostal myalgia, only a sign of muscular weakness, and in itself of no serious import. In most, however, the pain is truly cardiac in character, and though, strictly speaking, myalgic also, yet as the debilitated muscle whose action is painful is the central organ of the circulation, it is of much more serious import; it is still perfectly curable, but probably only differs in degree from the suffocative breast pang which has so many sad and fearful memories attached to it. Cardiac pain of this kind is obviously associated with imperfect nutrition, and when this imperfect nutrition is concomitant with an organic cause, the



pain is apt to be more permanent and severe ; and if to any physical cause of mal-nutrition we have superadded some physical source of obstruction necessitating a more than usually powerful action to overcome it, then we have a strain thrown upon the cardiac muscle which it is physically unfit, for and which it resents, or, to speak more correctly, indicates, by the pain which attends its action. Hence there are few cases of cardiac disease which in their uncompensated stages are unattended by pain. This pain is more or less constant ; it may vary in degree, and it does so vary, but it is seldom absent, and its cessation is a tolerably certain sign that compensation is more or less perfectly restored.

We have also a paroxysmal form of cardiac pain which is not associated, or, at all events, not usually recognised as associated, with any detectable form of cardiac lesion. In rare instances the victim of this affection is woke up from sleep by the first attack, which, repeatedly recurring, cuts him off in a few hours, as in the case of the late Dr Arnold of Rugby.\* In most cases the attacks are repeated, and they often last for years, and, with our modern methods of treatment, I believe they are not likely to be so suddenly fatal as in the case just mentioned. Generally the first attack comes on as the patient is ascending some slight acclivity, or making some trifling exertion, possibly after a meal. He is suddenly pulled up by an excruciating pain shooting through the lower part of his sternum to his back-bone, often accompanied by a constrictive feeling, as if his chest was grasped by a mailed hand ; the pain may remain localized, or it may shoot towards the shoulder down the left arm by preference, or down both arms. These are its most usual courses, but it may also occasionally shoot down the abdominal and lumbar nerves. The patient is at once brought to a standstill ; he fears even to breathe, but if he chooses to make the effort, he can breathe freely enough ; he feels a sensation of impending death, and a ghastly paleness overspreads his countenance. The pulse may intermit, or be

\* Latham on *Diseases of the Heart*, 1846, vol. ii. p. 373.

feeble or irregular, but it is sometimes—especially if the angina be uncomplicated by any other disease—quite regular throughout the whole of the paroxysm. After a few seconds the pain ceases as suddenly as it came on, and the patient finds himself as he was, puzzled to know what has happened to him, and terrified at the prospect of a recurrence of the attack. There is in this seizure nothing pulmonary; the air enters freely into the lungs if the patient has the courage to breathe, and full inflation of the lungs has no influence upon the attack. It has no apparent connection with cardiac or spasmodic asthma, neither does it arise from cardiac strain, or at least from any immediate and recognisable cause of strain. At first it seems to originate in some trifling exertion, or in some emotional excitement of the heart's action, but presently not even these exciting causes are necessary, and now and then, as we have seen, even the primary attack is without any such provocative, the patient waking from sleep in a paroxysm of anginous pain. In most cases it is not till after several and often numerous attacks, brought on by trifling exertions, that the disease arrives at such a pitch as to occur when the patient is asleep or at perfect rest, but sooner or later it reaches this stage.

Neither excruciating nor commanding are words strong enough to express the character of the pain in this affection; it seems to be something appalling, it unnerves the strongest mind, and death itself seems preferable to the repetition of a similar seizure.

Should death be the result of such an attack, the heart is found loose, flabby, and uncontracted—not exactly the condition in which one would expect to find it were death due to spasm, as Latham, Heberden, and the older writers imagined, but very much as it ought to be were death due to inhibitory paralysis, as Parry, Stokes, and Walshe\* have

\* Anstie seems also to lean to this view, *vide Neuralgia and its Counterfeits*, London, 1871, p. 73. Dr Moinet likewise adopts the idea of paralysis in contradistinction to spasm, *vide Treatise on the Causes of Heart Disease*, 1872,

supposed. And the history of the mode of death in this disease corresponds thereto, for in such cases death is not usually instantaneous, as would be the result of a suddenly fatal spasm of the heart, but commonly occurs from a gradual sinking of the aortic pressure, the result of an equally gradual diminution of the heart's force, the pulse getting feebler by degrees until it ceases, and never passing at once from its ordinary force to a full stop, as would be the case in sudden cardiac spasm. Moreover, the pathology of angina pectoris as a neuralgia of the cardiac nerves is in accordance with this view of the cause of death, for we know that any sudden and violent pain produces sickness, faintness, and depression of the heart's action; and we also know that whatever produces depression of function in the fibres coming from the posterior root of a spinal nerve, and as its result pain or neuralgia, produces also depression of function of the motor fibres coming from the anterior root of the same nerve, and as its result subparalysis of the parts to which they are distributed.\* Hence we have in angina pectoris two distinct sources of depression of the cardiac action: first, we have the directly depressing influence of a pain, the most acute and severe which the human frame can experience; and second, we have the action on the cardiac motor ganglia of the same cause which, acting on the sensitive nerves, gives rise to this excruciating agony, and we cannot but suppose that as a rule the functional depression of the motor nerves is not much less than that of the sensitive ones, that is, that the subparalysis of motion must bear some proportionate relation to the acuteness of the pain, which is the index of the functional depression of the nerves of sensation. This, however, is a rule to which there must be some exceptions; even in strictly spinal nerves the pain is often very

p. 102. Eichwald, "Ueber das wesen der Stenokardie," &c. *Wurzburger Medizinische Zeitschrift*, IV. Bd. 4 heft., 1863, gives a good historical *résumé*, and regards angina as very often a reflex neurosis which is fatal by arrest of the heart in diastole.

\* Anstie, *op. cit.* p. 6; Van der Kolk *On the Spinal Cord*, &c., New Syd. Soc. Trans., 1859, p. 7.



painful sensation in the left breast, we must never forget that we have to do with a cardiac lesion which requires special treatment to cure it quickly and well. Between the slighter forms of mere cardiac uneasiness and the distinctly paroxysmal variety attended by severe precordial pain, shooting in various directions, but most commonly up the chest and down one or both arms, and accompanied by an overwhelming sense of impending dissolution, and all the signs of serious nervous shock, there are many intermediate grades of severity. And though we might theoretically desire to exclude from the category cases of tumultuous and forcible heart-beat with lancinating pain, as apparently inconsistent with the idea of the subparalytic character of angina, yet this disease may begin in this way. Though I myself would not therefore apply the term angina to cases presenting these symptoms, yet true angina may develop out of them, as the following most interesting case abundantly proves :—

CASE XXVII.—A. S., a male, æt. 24, admitted to Ward V. 6th October 1877, complaining of palpitation and pain in the precordial region. The patient stated that he had suffered in this way for fourteen weeks, and that the pain was sometimes a mere uneasiness, and at others more acute. On admission, the patient presented a somewhat anxious expression, and was found to be well nourished, all his organs being healthy with the exception of the heart, which beat rapidly (120 per minute) and in a somewhat tumultuous manner; the radial pulse was quite regular. On auscultation, the first sound at the apex was impure, the pulmonary second markedly accentuated, and a faint diastolic murmur was audible over the aorta at mid-sternum. The case was at once recognised as a serious inflammatory affection of the heart or ascending aorta, but whether it was endocarditis, myocarditis, or endarteritis, the symptoms were not distinctive enough to decide. The treatment consisted in the administration of full doses (15 grains) of iodide of potass in a bitter infusion three times a-day, with perfect rest in bed, and an unstimulating diet. Under this

treatment his heart quieted down, but the pain increased, and became localised as a constant pain in the *scrobiculus cordis*, unaffected by pressure. This extension of the pain to the epigastrium was at first regarded as due to the action of the iodide on the stomach; but finding that it recurred in a distinctly paroxysmal manner, and that the pulse became rapid and feeble both during the paroxysm and for some little time after, the diagnosis was at once completed, and the affection stated in all probability to be an acute endarteritis of the cardiac end of the aorta, implicating the openings of the coronary arteries, with consecutive (febrile) dilatation of the heart. At first inhalations of nitrite of amyl gave great relief, the paroxysms lasting from five to fifteen minutes. But in the final attack, which commenced about four o'clock on the morning of the 24th of October, and lasted for about two hours, the amyl was of no use, and the only relief obtained from the intense agony was from chloroform inhalations.

At the autopsy on 25th October the body was found to be well formed and fairly muscular. Rigor mortis and post mortem lividity well marked. Thorax: about six ounces of clear serum in each pleural sac, and about two ounces of similar fluid in the pericardium. The blood was remarkably fluid. The heart weighed thirteen ounces, the ventricular cavities were slightly dilated, and their walls slightly hypertrophied. The mitral orifice was enlarged, admitting four fingers; cone diameter, 1.6. The cusps were natural. The tricuspid orifice was also enlarged, admitting six fingers; cone diameter, 1.9. In the wall of the aorta, immediately beyond the aortic cusps, there was a ring of atheromatous thickening which involved the whole circumference of the vessel at and a little beyond the sinuses of Valsalva. In this situation the tunica intima had grown to twice or thrice its natural thickness, and presented a clear, translucent aspect, being only here and there affected with points of fatty degeneration. The openings of the two coronary arteries lay in the midst of this atheromatous area, and were both so



extremely contracted as barely to admit the point of an ordinary surgical probe. The aortic valve allowed water to leak through it slightly, but it might be said to be practically competent. The cusps were slightly thickened at their free margins, and above the *corpora aurantii*. The muscular substance of the heart was everywhere of good colour and consistence, and on microscopic examination presented no abnormality except the presence of a considerable number of reddish brown pigment granules in some of the fibres. All the other organs of the body were perfectly healthy, but somewhat congested.\* In this most interesting and probably unique case you will observe that we have a fatal cardiac lesion coincident with a practically healthy heart. The slight dilatation, slight hypertrophy, and trifling leakage through two orifices were nothing more than we may find in any spanæmic heart, especially when febrile excitement coexists. Similar conditions are to be found in hundreds who make perfectly good recoveries. The fatal lesion was evidently the blocking of the coronaries; this was what was found,\* and this was precisely the lesion which had been predicted to be the most probable one. This opinion was based on the obviously causal connection between the acute attack and the angina in this case, and on the fact that in by far the larger number of cases angina seems to depend upon some interference with the blood supply to the walls of the heart itself, and consequently to the intra-cardiac nerves. In a very large experience of angina, including over a dozen cases known to have been fatal, I have never failed to detect indications of defective blood supply to the heart, and in the only three dissections I have had this view has been abundantly confirmed. The case just narrated supports this opinion, as indeed almost all those recorded also do, even the celebrated case of Dr Arnold being no exception. For though his coronary artery—he had but one—was neither

\* Condensed from the Pathological Records of the Edinburgh Royal Infirmary. A chromo-lithograph of this heart forms the frontispiece to this volume.



diseased nor obstructed, it is stated that, "considering the size of the heart, it appeared to be of small dimensions, and with some difficulty admitted a small director" (Latham); while the thin, soft, flabby texture of the heart sufficiently testified to the inadequacy of its feeding power. That is the true cause of angina; so long as the coronary arteries are able to feed the heart, it matters not what their structure is, we have no angina. Whenever from any cause the blood supply to the heart is insufficient, then we are liable to have angina, and in all such cases we have more or less of it. Pressure on the cardiac nerves in some part of their intra-thoracic course is an occasional cause of angina, but there is every reason to believe that even in this case it is produced by direct or reflex interference with the intra-cardiac circulation. The sequence of events in ordinary cases of angina seems to be, first of all imperfect nutrition of the cardiac muscle, associated with various uneasy or painful sensations, and generally (always?\*) accompanied by the early physical signs of dilatation, usually with some hypertrophy. Next we have paroxysmal attacks of pain occurring when the heart is called upon for extra exertion, especially when a weak pneumogastric nerve is irritated by a distended stomach. At first these attacks only occur when the patient is debilitated from any cause, and his cardiac power thus temporarily impaired; in these circumstances improvement in health is followed by cessation of the angina, temporarily or permanently. By and by, as the nutrition of the heart becomes more impaired, the attacks are more readily brought on; the most trifling excitement of the heart's action, whether induced by exertion or by irritation of the terminal branches of the pneumogastric in the stomach, suffices to induce a paroxysm of pain; and in some cases at last the

\* I have never seen a case of angina in which these signs were not present. At present I am inclined to lay some stress upon the absence or presence of these signs of cardiac dilatation as an indication of the dependence of the pain on substernal aneurism, or of its strictly cardiac origin. But my experience as yet is insufficient to enable me to assign more than a moderate probability to this assumption.

ordinary action of the heart is painful, aggravated by continually recurring paroxysms of greater severity, which wax and wane in that inscrutable fashion so common in other neuralgias.\* Death occurs from asystole or rupture, sometimes during an attack, more often, perhaps, during a painless interval.

Such seems to be the history of all ordinary forms of angina. In traumatic angina the case is different. Here we have an individual apparently in the most perfect health, who meets with some trifling accident, and is at once plunged into a series of recurrent paroxysms of the most severe cardiac pain, which ere long terminate in death. The only case of this kind which has occurred to me was the following:—

CASE XXVIII.—J. L., a married woman, aged 50, stout, healthy, and who had passed through her life without an ache or a pain except those incident to maternity, slipped and fell on the street in the beginning of January. Being rather heavy, she was considerably shaken, but apparently not otherwise injured. By and by, however, anginal paroxysms set in, and continued gradually to increase in severity. There was nothing abnormal to be detected about her heart, but she died suddenly in a paroxysm about the middle of March of the same year. Unfortunately I was unable to obtain a post mortem examination; but from the sudden onset of the angina, its obvious dependence on the fall, and the resemblance of the most prominent symptoms to those of substernal aneurism, there is every reason to believe that the middle coat of the aorta was fissured transversely just above its cardiac origin at the time of the fall, that the angina was caused by pressure on some of the branches of the cardiac plexus by the gradually increasing aneurism thus formed, and that death was caused by this aneurism bursting into the pericardium.

The two cases just narrated have been the only cases in my

\* In regard to the periodicity frequently presented by neuralgias depending upon persistent grave organic lesions, *vide* Trousseau's *Clinical Medicine*, New Sydenham Society's edition, vol. 1. p. 598.



own experience who have died during a paroxysm, and I am inclined to believe that with improved methods of treatment this will be an increasingly rare mode of death. All the others have died from asystole brought on in various ways during the painless interval. One, after many years' freedom from pain, died quietly after suffering for some time from gradually increasing dropsy and other signs of a dilated heart. Another, who during several years suffered from many comparatively slight attacks of angina, lunched cheerfully with some friends, walked with apparent ease to a railway station but a short distance off, sat down, and died. Others, after suffering more or less intensely at intervals for years, died more or less suddenly without giving any indication of suffering. And one well-known literary man I myself saw die from ingravescient asystole about a week after his last paroxysm. He had for long suffered from angina, with all the signs of a weak, dilated heart, due to arterial atherosclerosis, and I had brought him safely through two most severe attacks with a comparatively painless interval of a year between them. Subsequent to the last attack he had been confined to bed with symptoms of pulmonary oedema, which is so common a result of a severe paroxysm. At last, when apparently fairly convalescent, he obtained permission from his medical attendant to rise from bed, and while dressing his weak heart failed and ingravescient asystole set in. There was no pain. When I reached his apartment he said, "Doctor, this is very different from anything I have had before," and he died quietly after drinking about half a glass of brandy given him in the hope of stimulating the heart to more vigorous contraction. The whole act of dying occupied about half an hour. At the post-mortem examination "both coronaries were found atherosclerosed and obstructed, and in the substance of the left ventricle there was an elongated patch of advanced fatty degeneration. The limits of the patch were well defined, and the appearance presented bore a considerable resemblance to that of a hæmorrhagic infarction



which had undergone fatty degeneration."\* The heart itself was somewhat dilated.

Of course any deficiency in the blood supply to the cardiac walls must injuriously affect the intra-cardiac motor ganglia, and thus we have the element of danger introduced. When an anginous heart retains a fair amount of reserve power, and the pain is of short duration, the danger may not be great, and, as occasionally happens, especially in cases complicated with aortic regurgitation, the sufferer can sometimes by a voluntary effort call upon this reserve power, flush his heart with blood, and overcome his breast pang. I have already related one such case (vide page 274), and I can recall several others; but the experiment is dangerous and failure is sudden death.† If, however, the reserve power is feeble, then the danger is probably commensurate with the severity and duration of the pain, and it is a matter of paramount necessity to relieve this as speedily as possible.

Our forefathers had nothing to trust to but the external application of cutaneous irritants, and the exhibition of stimulants and narcotics by the mouth; a vain hope, when moments are precious, and to the time needful for absorption—twenty minutes in the most favourable circumstances—was superadded the further delay occasioned by a failing circulation. Modern discoveries have changed all this; we can now by inhalation thoroughly narcotize a patient in a few seconds, and by means of hypodermic injection secure in ten minutes a painless unconsciousness which will last for many hours.

Foremost among all our modern appliances for the relief of this dreadful breast pang we must place the nitrite of amyl; it is perfectly safe, and may be entrusted to the patient with the certainty that he will not injure himself by its use; in all slighter attacks it serves to give perfect relief, and in more

\* I quote from a letter received from Dr Wyllie, at that time pathologist to the Royal Infirmary, who made the dissection, the full report having been unfortunately lost. The specimen is in the University Museum.

† Forbes, *Cyclopædia of Practical Medicine*, vol. i. p. 94, also mentions similar cases.

severe paroxysms it alleviates even when it cannot completely remove the pain. It flushes the face, quickens the heart-beat, and has been experimentally found to lower the blood-pressure in animals to whom it has been administered. It was originally employed by Dr Lauder Brunton in the treatment of angina, on the supposition that this depends upon increased intra-arterial blood-pressure. I quite agree that in all cases of angina the blood-pressure is probably always above the normal, but that it is abnormally increased at the moment of seizure has not yet been proved, and, so far as I know, is incapable of proof. The supposition of Dr Brunton that the case is so rests solely on a single sphygmographic tracing from the radial artery; but a pulse-trace only represents the local movement of the arterial wall, and for many obvious reasons can never be accepted as a correct indication of the intra-arterial blood-pressure. Further, if we accept the face-flushing as a proof of lowering of the blood-pressure, then I am in a position to state that two specimens of nitrite of amyl will flush the face in apparently the same degree, yet only one of these will relieve the pain. The specimen which relieves the pain is one which has been freshly prepared, or which has been kept in a hermetically sealed capsule. The other specimen, which does not relieve pain, has been kept for some time in an ordinary stoppered bottle. I make these statements from a large experience of the use of the nitrite of amyl, and chiefly base them on two cases, both of whom suffered for years from terrible angina. One of these cases had a loud, musical, diastolic aortic murmur, and he never felt well unless his wife could hear this murmur across the dinner table, a condition indicating, of course, a much greater blood-pressure than when the murmur ceased to be audible and his sufferings began. During the last few years of his life he used many pounds of the nitrite of amyl, having it constantly with him, and inhaling it when required. His face was always fully flushed, and a certain amount of relief obtained, but this relief was only rapid and complete when the specimen employed



was perfectly freshly prepared. The second case was somewhat similar. The sufferer was a medical man, and he used to soak his pocket handkerchief in the amyl and go to sleep with it on his face. The conclusion I have arrived at from these facts is, that the relief to the pain of angina is obtained, not from lowering of the blood-pressure, but from the action of a volatile narcotic, which gradually escapes from the amyl when kept, unless it is enclosed in hermetically sealed glass capsules. As these are now readily obtained, we possess a remedy which can be safely entrusted to the patient, and which is certain to give relief in all ordinary attacks.

When the attack is a severe one, the amyl fails to give relief, however freshly it may have been prepared; of this I have been assured by many sufferers, and have myself repeatedly had occasion to observe it. Then our only resource lies in chloroform, which can only exceptionally be entrusted to the patient, but ought always, if possible, to be given by a medical man. It must be given freely, so as completely to narcotize the patient; and, when so given, I myself have not yet seen any case which was not relieved, though I have seen several in which the relief was not permanent enough to place the patient in safety. In these cases I have had recourse to the subcutaneous injection of morphia, using by preference Squire's solution of the bimeconate, of which I have injected half a drachm into each arm, without removing the clothes or in any way disturbing the patient. This, as yet, has never failed me: the chloroform sleep has passed into the morphia sleep, from which the patient has woken up some hours subsequently, free from pain, but exhausted, as we can readily suppose, and usually with some cedema of the lungs. You see, then, that I have no dread of chloroform in these cases, and by no means homologate Anstie's statement, that "the only kind of chloroform inhalation which would be useful in such cases would be that in which a carefully measured small dose of a weakly impregnated atmosphere should be inhaled, and without large



experience in the administration of chloroform the practitioner will be unable to secure this effect with certainty; and the effect of a *powerfully* charged atmosphere, breathed only once or twice even, would be instantaneously fatal."\* You have seldom an opportunity of seeing the usefulness of chloroform in angina pectoris in the wards, but you have all at least occasional opportunities of seeing its beneficial action in other kinds of cardiac pain; and what is safe enough in aortic regurgitation for instance, cannot be dangerous in angina pectoris. Many years ago I knew an individual who was forbidden to take chloroform on account of a cardiac valvular lesion under which she laboured, and yet for long she secretly indulged in chloroform intoxication without any fatal result. So far from being unsafe in cardiac disease, it is often of the greatest use in these cases; it not only relieves pain, but regulates the circulation, now and then bringing the pulse back to the wrist, whence it had apparently fled for ever. In peritonitis this effect is occasionally quite remarkable. I well remember one case of extreme cardiac pain and dyspnoea in a patient almost moribund from cardiac disease, and who did die only a few days subsequently, yet in her the immediate effect of chloroform inhalation was to restore the pulse to her wrist, to enable her to breathe more freely, and in a few seconds, instead of being black in the face, pulseless, and gasping partly from pain, and partly from extreme dyspnoea, she became quiet, natural in appearance, and in a short time was able to lie down and rest. But, you may say, in angina the heart is almost universally flabby and fatty; is chloroform not dangerous when we have a fatty heart? The next case which I shall relate will be the best reply to this question. First, let me say that I doubt the possibility of diagnosing a fatty heart. We may suspect its existence, because the physical signs seem to warrant the supposition, while the conditions present are apparently favourable to its development. Thus there may have been a long-persistent spanæmic condition of the blood,

\* *Op. cit.* p. 80.

or there may be a state of general (pernicious) anæmia, or there may be reasons for suspecting a purely cardiac anæmia from local causes, such as an atherosed and obstructed state of the coronary arteries, or an overgrown hypertrophy of the cardiac muscle, which has got beyond the feeding powers of these vessels. For fatty degeneration of the cardiac muscle is the result of mal-nutrition, and seems never to be found apart from one or other of these conditions. But little acquaintance with pathology is, however, requisite to teach us that these conditions are not all of them easily and certainly recognisable, and that even when present fatty degeneration is not an invariable concomitant of any of them. Though, therefore, our suspicions may occasionally be right, they may more often be wrong. The signs of cardiac debility upon which we base our suspicions are much more commonly due to dilatation than to fatty degeneration, in spite of the possible co-existence of an arcus senilis. Besides, there are many cases of actual fatty degeneration in which there have been no faintings, cardiac asthma, feebleness of the pulse or of the cardiac impulse, no yellowness or pastiness of the complexion, and no arcus senilis—in fact, all the symptoms and signs connected with the heart have either been those of perfect health, or at all events they have not been such as are generally supposed to indicate fatty heart, yet the heart has been markedly fatty.\* I may refer to the case of Bridget Henry, who died from chloroform in the Cincinnati Hospital, U.S., 13th October 1870,† as a well-known example of the conjunction of a normal impulse with a fatty heart; but indeed of this we could scarcely have a more striking instance than the case of Mrs Tait, which I will presently relate to you. No doubt the

\* While this is passing through the press this statement has received a most forcible illustration in the death, from rupture of a fatty heart, of one of our best known and most esteemed medical practitioners, whose healthy appearance and great vigour both of body and mind were entirely opposed to all the more generally received ideas in regard to this form of degeneration.

† *Chloroform Deaths*, by W. W. Dawson, M.D., Surgeon to the Cincinnati Hospital, 1871. Printed by Robert Clarke & Co., Cin.

rapid and powerful action of chloroform renders it a very dangerous agent in incautious hands, and some diseased states of the heart, of which an enfeebled and anæmic condition are the chief, render it more sensitive to the action of chloroform, and more liable to be fatally overpowered by an overdose; but I know of no diseased condition which should deter us from its cautious employment when that is otherwise indicated, as I hold it imperatively to be in certain cases of angina pectoris, for what we desire to do in them can only be done by means of chloroform. I do not say *tuto, cito, et jucunde*, because the superlatives of these adverbs are more applicable than their simple positives. In a disease possessing such a pathological history as angina pectoris, the one great object is to free the cardiac nerves from the depressing influence of pain which gives rise to that subparalysis in which the danger lies. This we can only do by narcotizing the nerve centres through which this action takes place, and so setting them free from all those influences which tend to depress the heart's action. The immediate result is a sensation of relief from pain, greater force and freedom of the heart's action, and a fuller pulse.\*

Sulphuric ether has long been used with a similar intent; it is an admirable narcotic, and the chief objection to its use is that it is not rapid enough, taking always some minutes to bring the patient fully under its influence. Chloroform acts more quickly, even more effectually, and is perfectly safe. It is not always necessary for the medical man to administer it, though in some cases it is so. All that we require is to insure that the patient shall only have a moderate dose, and this we manage by giving him a chloroform smelling-bottle, the fluid

\* In his work on *The Bearings of Chronic Diseases of the Heart upon Pregnancy, Parturition, and Childbed*, London, 1878, Dr Macdonald says, *apropos* of a primipara labouring under aortic insufficiency, "The patient looked pale . . . and complained that she felt ready to faint with every pain." Though besought to give chloroform, he hesitated, but yielded at last, and then found that "under its employment the pulse became stronger and steadier, instead of feebler and more irregular, as I feared it might."—*Vide* p. 147.



being poured over a piece of sponge, so that it cannot spill; this smelling-bottle he is told to hold to his nose, and to breathe as deeply as possible. In this way relief is obtained in a few seconds, and so soon as the narcotic influence is produced the smelling-bottle drops, and with it rolls away all risk of any overdose. Mrs Tait, long a nurse in the Infirmary, and who died in Ward XIII. on 30th March 1871,\* was for the last few weeks of her life almost constantly under the influence of morphia or chloroform, or both, the morphia being injected hypodermically as soon as the chloroform narcosis was established, so that its soothing influence might come into play when that of the chloroform passed off. She was over eighty years of age, and had long suffered from angina pectoris, the paroxysms of which were latterly very severe, and with but short intervals between them. She died at last, as I have said, not suddenly, but gradually, worn out by her age and sufferings. After death the aorta was found dilated, the orifice of the middle coronary artery—there were three in her heart—almost entirely blocked up by atheromatous deposit, and her heart not only thin walled and somewhat dilated, but of a pale yellowish tint, soft, and thoroughly fatty; no muscular fibres could be more completely degenerated; yet chloroform produced in her no dangerous symptoms, and, far from shortening her days, seemed to prolong them. I may also add that her apex beat was tolerably firm to the last.

But however satisfactory our treatment of the paroxysm is, the treatment during the intermission is quite as important, and is often attended by even more striking results. From the pathology of this disease already given you will understand that during the intermission I use every endeavour to improve the patient's general health, and especially to tone up his heart. To this end he must be warned to avoid every source of excitement, to take perfect rest in a mild and equable climate, where he ought to be much in the open air, driving or sitting, but not walking. His diet must be so regulated as to consist

\* Margaret Tait, admitted 3d January, died 30th March, 1871.

of the blandest, most nutritious, and unstimulating foods, avoiding everything likely to prove difficult of digestion or give rise to flatulence, and being particularly careful as to stimulants, the action of which is ultimately to weaken the heart, and the more stimulating such drinks are, and the greater the amount partaken, so much more rapidly is this result attained. Of course the whole system must be carefully attended to, and acid tonics, pepsine, mild laxatives, or any other general remedies given that may seem to be required. But as all secondary functions depend for their perfect discharge very much upon the condition of the great central organ, so our best mode of improving the gastric or hepatic functions will always be to improve the heart. To that end we have as yet only two remedies of any importance: these are arsenic and digitalis. Very shortly after Fowler introduced his tasteless ague drop, arsenic was employed experimentally in a great many diseases, angina pectoris amongst the rest, and in several cases it was found to be successful.\* Since then it has been often used in such cases. Anstie has declared it to be "an invaluable remedy in cardiac neuralgia," as well as "the most important prophylactic tonic"† we can employ in these cases, and my own experience is quite to the same effect. Arsenic is indispensable in all forms of weak heart accompanied by pain. It is useful in all such cases, and in many it is quite successful in putting a stop to angina. Several cases have occurred to me in which arsenic alone has removed angina after a few weeks' treatment not only temporarily, but permanently. The ordinary dose is from three to five minims twice a day after food, but the dose may sometimes be advantageously pushed till slight physiological symptoms appear, and thereafter continued so long as desired in a dose just short of that needful to produce these effects. Its mode of action is somewhat obscure. Besides being a good general tonic, as well as a special tonic to the heart, it

\* Forbes, *Cyclopædia of Practical Medicine*, vol. i. p. 95.

† *Op. cit.* pp. 78, 79.



seems also to exercise some modifying influence on the nerves which renders them less liable to pain in spite of a continually advancing degeneration. But however it may act, arsenic is a drug well worthy of confidence in the treatment of angina, and associated with iron and strychnia it forms a combination specially valuable in all cardiac neuroses. One great difficulty in the administration of arsenic lies in its tendency to irritate the bowels of some patients; this may be overcome by the addition of opium, or by the diminution of the dose, for it not infrequently happens that a constitution sensitive to the injurious action of a drug is also sensitive to its curative action. I have frequently found this to be the case with arsenic, and it is always worth remembering. I distinctly remember one gentleman specially sensitive to the action of all drugs, but particularly to that of arsenic, who could not bear it in larger doses than one milligramme of arsenious acid daily (0.015 gr.); two milligrammes gave him discomfort, one was well borne. He was not aware in what the evil effects of the drug consisted, nor did he know what good I expected from its use, yet in about a fortnight he said, "I feel my breathing easier now; my heart is steadier, and I can go upstairs better than for many years." No better results could have been obtained from larger doses, and, indeed, from his sensitiveness, had these been persisted in, the drug would assuredly have had to be entirely given up. From my belief in the connexion between defective nutrition of the heart and cardiac pain you will readily understand that I put considerable faith in the use of digitalis in these cases, in small tonic doses repeated night and morning. Ten minims of the mixture of digitalis, or one granule of Nativelle's digitaline, is an ample dose, and I have seen nothing but good result from the use of this drug. One granule of digitaline night and morning, with arsenic, strychnia, and iron twice a-day after food, is a sort of model treatment, for such cases, and this treatment, coupled with nourishing, unstimulating food, abundant rest and fresh air in a mild and



equable climate, is often attended by the happiest results in those cases susceptible of improvement, which are, in truth, by no means of infrequent occurrence.\*

\* I may add that I have used Glonoine (Nitro-Glycerine) without any benefit in the treatment of angina. I have employed the nitrite of amyl hypodermically and also internally, but have found these methods inferior to inhalation in angina. I have, however, found the internal use of the nitrite of amyl useful in one of those rare cases (of which I have only seen two) in which some vaso-motor disturbance is associated with a permanent and most distressing feeling of cold. The patient, a joiner to trade, could not handle his tools because they abstracted too rapidly the little heat he could produce, and seriously aggravated his condition. He subsequently died from what appeared to be a universal chilblain, passing into unhealthy suppuration.

## LECTURE XIII.

ON PERICARDITIS, MYOCARDITIS, AND CARDIAC HYPERTROPHY,  
APART FROM VALVULAR DISEASE.

GENTLEMEN,—Though acute articular rheumatism is by no means a rare disease with us, yet rheumatic pericarditis, worthy to be so called, is almost unknown. This is all the more remarkable because our largest ward is a female one, and you are, of course, aware that rheumatic pericarditis is more prone to attack young and weakly persons, especially females, than any other class of patients. Were it not that rheumatic pericarditis is usually an early and rarely a late complication—occasionally even preceding the joint affection—we might be inclined to attribute its rarity to the success of our treatment; but however much may be due to that, the fact that few of our patients come under treatment before the fourth or fifth day, coupled with the rarity of severe pericarditis, seems to indicate that rheumatic pericarditis is really rarer in Edinburgh than elsewhere, or at all events much less severe, a probability which has also suggested itself to Dr Gairdner.\*

The invariable treatment for acute rheumatism in my ward, as you very well know, is first of all to clothe the patients in flannel and bed them in blankets, a mode of treatment which, though comparatively recently recommended anew by Dr King Chambers, is of very old date in this Infirmary, as you will find by a reference to Dr Cullen's "Clinical Lectures" delivered here in the end of last century, a MS. report of

\* *Ed. Med. Jour.* Jan. 1861, p. 630.

which is to be found in the library of our Royal College of Physicians, and in it you will find this procedure very strongly inculcated as a most important part of the treatment of acute rheumatism. Along with this I give my patients—not Doyer's powder, as Cullen did—but the tincture of the *actæa racemosa*—the American Bugwort—in doses of one drachm every two, three, or four hours, according to the severity of the symptoms; I also give a full opiate or dose of chloral at bedtime, and wrap each affected joint in cotton wool, putting a large sheet of carded cotton over the chest. Under this treatment recovery is not usually long delayed; many convalesce within the week, and the disease seldom lasts longer than two, or at the most three, weeks. In exceptionally severe cases, when the disease is obstinate, and febrile excitement high, I have recourse to aconite in full doses, carefully watched, but this has rarely been required, and has always been successful. When the fever ceases and the joint ailment continues I put the patient upon half drachm doses of the iodide of potassium three times a day, or upon full doses of arsenic, or occasionally both are combined; the result has hitherto been satisfactory.

[Very shortly after the foregoing was published, the bark of the willow tree, in its modern analogues of salicin and the salicylates, came to the front as almost a specific for rheumatism, and in my own practice has with rare exceptions entirely displaced all other treatments for this disease. Willow bark is said to have been known to the Hottentots from time immemorial\* as a remedy for rheumatism; it has certainly been known to medicine as a febrifuge or antipyretic at least since the middle of last century,† while the febrifuge properties of its active principle salicin have been known for the last fifty years.‡ But the use of salicin and the salicylates as a modern specific for acute rheumatism, dates only about six

\* *Vide* Dr Enson's letter, *Lancet*, 1876, vol. i. p. 910.

† *Disp. ii. de cortice Salicis cortici Peruviano substituendo*, J. W. Guenz, Lipsie, 1770.

‡ *Enai sur la Salicine et sur son emploi dans les Fièvres Intermittentes*. J.B. Blaincourt, Paris, 1830.



years back, and its employment has quite revolutionised the treatment of that disease, so that instead of one week being an exceptionally short period for such patients to convalesce in, it is now an unusually long time for the disease to last. In spite, however, of the success which attends this treatment, so far as shortening the duration of the fever is concerned, it is no true specific. It does prevent the occurrence of such untoward accidents as fatal hyperpyrexia, of which one rapidly fatal example has occurred in a patient under my own care, while apparently going on favourably under the salicin treatment. Her temperature at night was only  $102^{\circ}$ ; she appeared no worse in the early morning and made no complaints, but on the nurse going to her at nine o'clock she was found unconscious, perspiring freely, and with a temperature of  $107^{\circ}$ . She died at half-past twelve, with a temperature of  $111^{\circ}$  F., which persisted for an hour after death. On post mortem examination no lesion was found to account for this remarkable and fatal rise of temperature.\* Neither has the salicin treatment any apparent effect in diminishing the risk of ulterior affections of the heart; yet my own belief is that the marked shortening of the duration of the acute stage of this disease, produced by salicin, cannot but have an important influence in preventing secondary cardiac disease, but that in the meantime this is masked by the inappreciable but not less important results of previous attacks, and will not be fully recognised until salicin has been generally employed for many years. In regard to acute cardiac affections of rheumatic origin, these as a rule occur early in the disease, before treatment has been commenced, and cannot well be prevented by anything short of a prophylaxis so perfect as completely to extinguish the disease. salicylic acid and the salicylate of soda were first used in the treatment of acute rheumatism by Traube, Riess, and Stricker of Berlin in 1875,† and in 1876‡ Dr MacLagan, now of London,

\* *Vide* "Comments on a Case of Hyperpyrexia," by Theodore Cash, M.B. &c., *Ed. Med. Jour.* Sept. 1878, p. 234.

† *Vide Berliner Klinische Wochenschrift*, 1875, p. 673, and 1876, p. 1, &c.

‡ *Lancet*, 1876, vol. i. p. 342.

recommended salicin as a specific for that disease. I have always myself employed salicin, as it seems safer, pleasanter to take, and perfectly efficacious. One ounce of salicin is the smallest quantity with which we can expect to make any impression on an acute rheumatic attack, and the sooner we get this quantity thrown into the system the better for the patient. Maclagan recommends thirty grains of salicin to be given every hour till one ounce is taken, when the symptoms will be found to be much modified if not completely checked, relief to the pain being one of the earliest indications of improvement. Should there be any considerable remission of the symptoms before the first ounce is entirely consumed, the interval between the succeeding doses may be increased to two or three hours. To complete the cure a second ounce is to be given in similar doses at longer intervals, and it is often advisable to give a third ounce in half drachm doses three times a-day, to ensure complete and perfect convalescence.\* This treatment never depresses, but is strictly tonic as well as curative, and the occurrence of either endocarditis or of pericarditis does not in the least contra-indicate its employment.]

I have not paid special attention to the statistics of this disease, but I know that I have treated during the last eight years over 2000 cases of general disease in this Infirmary, of whom nearly 100 were cases of acute rheumatism; some of the ward journals have gone amissing, but I have records of 1968 cases of disease, 70 of which were cases of acute rheumatism, without one fatal case of rheumatic pericarditis, and I know that I have never had either in hospital or private practice one fatal case of rheumatic pericarditis, nor in fact have I ever had one case of this disease more serious than that which I am about to narrate.† I have already told you that rheumatic

\* *Vide* Maclagan, *Lancet*, 1879, vol. i. p. 875.

† I have not considered it worth while to tabulate my cases of rheumatism during the last five years, as during that period pericarditis as a complication of rheumatic fever has been not more frequent than formerly—always slight, and never fatal.



pericarditis usually arises early in the disease, and that our patients rarely come in till they have been some days ill, you will not therefore be surprised to learn that the origin of pericarditic symptoms subsequent to admission is of rare occurrence, but it is of more importance for you to know that even when these symptoms have been present on admission they have speedily abated. If we take cardiac pain accompanied by slight but still detectable effusion into the pericardium, or by friction about the base of the heart, accompanying acute rheumatism, as signs of rheumatic pericarditis, as they undoubtedly are, then I do not suppose that my percentage has been at all, if in any respect, less than that of my neighbours—25 to 30 per cent.—according to Bamberger—the mortality having been *nil*, in this respect also agreeing with Bamberger. Besides putting my rheumatic patients upon a milk diet, the only other care that has been exercised respecting them has been that, while daily examining them myself, and while affording, as you very well know, ample opportunity to all students to make themselves acquainted with the phenomena present, careless exposure of the patient has been as much as possible prevented, and all indiscriminate and unnecessary examination of the patients discouraged; in short, while in no respect neglecting your education, the welfare of the patient has ever been, as it ought to be, our paramount object; and in regard to this I have not been unmindful of Martial's epigram—

“Languēbam, sed tu comitatus protinus ad me,  
Venisti centum, Symmache, discipulis.  
Centum me tetigere manus aquilone gelatæ,  
Non habui febrem, Symmache, nunc habeo.”

Possibly this care may also have had something to do with the infrequency of serious pericarditis among the rheumatic patients under my care.

In not a few cases the rheumatic attack has been associated with cardiac disease of old standing, while in those free from



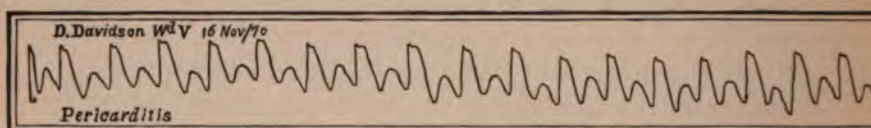
cardiac disease we have almost invariably been able to determine the existence of the auricular murmur which I have already told you is of such frequent occurrence in all febrile disorders, depending upon a slight degree of cardiac dilatation,\* and this has been occasionally, though rarely, accompanied by a murmur of similar origin audible in the mitral area. But the persistence of such a murmur, and its gradual transformation into one of distinct valvular lesion, has been of very exceptional occurrence. In by far the larger proportion of cases this murmur of dilatation has died completely out, and that of valvular lesion has only developed itself subsequently, sometimes gradually, without the occurrence of any definite symptoms, at others associated with repeated trifling subacute attacks of short duration, and at still others with the persistence of chronic wandering rheumatic pains, not always obviously affecting the heart. *Hæret lateri lethalis arundo* is an apothegm entirely applicable to rheumatism in all its forms, and if it finds its most apposite application in cases of rheumatic pericarditis, it finds it so rarely as to be of little consequence to the species, however important it may be to the individual himself. The following is the only serious case of this kind that has occurred to me during fourteen years' connection with this Infirmary.

CASE XXVII.†—David Davidson, a tailor, aged 24, admitted to Ward V. on 16th November 1870, complaining of pain in the chest and limbs, difficulty of breathing, and dry cough. Patient stated that he had always been healthy till about five years ago when he had slight rheumatic pains. The first attack commenced on 7th November, with pains in the joints of the lower extremities, but he continued at work till the 10th; on the 12th cardiac pain set in, and on the 16th he came into hospital. Both his parents were dead, his father had been rheumatic, his mother not. Upon admission he was found to have an anxious expression, his skin felt hot,\* and was

\* Lecture VI. p. 171.

† Case reported by Benjamin Tydd Hunter, Clin. Clerk.

covered with sour smelling perspiration, the temperature in the axilla was only  $99^{\circ}8$ , he had pain in the right knee-joint, but it was neither swollen nor red. The patient felt a burning pain in the præcordial region, the percussion dulness was normal, but a loud rough friction sound accompanied the movements of the heart, was loudest at the base, and with the systole, but it obscured both sounds. The pulse was 100, full, compressible, and markedly dicrotic, as is well shown by the



accompanying figure (fig. 20). The respirations were forty in the minute, and he had some cough with slight expectoration. Percussion was clear over both lungs, the respiration normal on the right side, and accompanied by coarse crepitation on the left side. His sleep was disturbed, and his appetite defective, his tongue was coated, dry, and brown in the centre and towards the back, white at the edges, bowels normal. Urine normal in quantity, acid, deep amber, spec. grav. 1026, containing a floating cloud of urates, and giving with nitric acid a deposit of nitrate of urea, chlorides normal. He was put upon infusion of digitalis and nitrate of potass, with chloral at night, and in two days the dicrotism of the pulse was no longer to be detected; it had fallen to 96. On 19th November the pulse was again 100, and the cardiac symptoms remaining unchanged, the rheumatism now passed into the joints of the middle finger of the left hand, his loins, and his left wrist, the joints being swollen, red, and painful. He was now put upon the tincture of the *actæa racemosa*, one drachm being given every three hours, a warm linseed poultice was kept constantly applied over the cardiac region. On 22d November the transverse dulness at the fourth rib measured six inches, it did not rise above the third rib, but was recognised as due to effusion within the pericardium, from the

disappearance of the cardiac apex beat and from the dulness passing from side to side as the patient was turned, first on the one side, and then on the other. On 24th November he had an attack of severe pleuritic pain, for which he had a hypodermic injection of morphia, and which resulted in effusion into both pleuritic cavities, the dulness extending as high as the eighth rib on both sides. On account of the persistence of this effusion, the other symptoms being moderated, he was ordered on 30th November

R. Potassii Iodidi  
Potassæ Bicarbonatis, āā ij  
Infusi Calumbæ, ℥vj

Sgr.—One tablespoonful in water three times a day.

After this he continued to improve. On 1st December, the thoracic dulness continuing the same, a friction sound was heard over the right base posteriorly, and next day over the same part on the left back, the dulness now only reaching to the upper border of the ninth rib. On 4th December a faint systolic murmur became audible in the mitral area. The patient continued to improve, the dulness over both lungs and heart gradually decreased. On 8th December all medication was stopped, and on 5th January 1871 he was sent to the Convalescent Hospital. I have frequently seen him since; he continues at work, but his mitral murmur is loud and unchanged, yet he suffers nothing from his cardiac ailment, and is in very good general health.\*

The diagnosis of pericarditis is now and then beset with some difficulties; for instance, the sound of friction most usually heard at the base of the heart, and which is generally distinct enough, often scrappy and well-marked, now and then creaky and obscure, occasionally simulates so closely the blowing sound of a valvular murmur as to be indistinguishable from such a murmur by the sound alone. I have most

\* January 1881.—This patient has been married for several years, he enjoys most excellent health in spite of the persistence of his mitral disease, and he has had no recurrence of his rheumatism.



frequently observed this in those chronic and latent forms of pericarditis which occasionally occur during the course of Bright's disease, but there never is any real difficulty in determining between a murmur and a frictional pseudo-murmur; in deciding this matter rhythm is of little importance, as a pseudo-murmur may be systolic or diastolic, or may occupy both times continuously, or the reverse—discontinuously and irregularly; but even valvular murmurs we know may vary in a similar manner, that is to say, they may be systolic or diastolic, or double, covering both systole and diastole, or they may occur irregularly. But a frictional pseudo-murmur has but rarely the same position of maximum intensity with the murmur which it simulates, at least that has been my experience, the only exception being the diastolic aortic murmur; pseudo-murmurs are also strictly localised to the cardiac area, and usually to a very small part of that, not being propagated out of their position of maximum intensity to any extent, and then only equally all round, and not in the definite lines of any special valvular murmur, while all the secondary results of the valvular lesion simulated are, of course, entirely wanting; moreover, it is only rarely that the natural sounds of the heart are lost in the murmur, though they are not infrequently obscured by it. When, therefore, we have over the cardiac area a murmur of any kind whatever that is not distinctly a sound of friction, we must always exhaust the possibilities of a valvular origin before we determine it to be of pericardiac origin, and the only real difficulty will be found to exist when such a pseudo-murmur occurs along with pre-existing valvular disease; then indeed the diagnosis may be almost impossible, but then too it is of comparatively little importance.

A friction sound heard over the cardiac area may be due to pleurisy alone, and may yet be audible during the cardiac action, when the respiration is stopped for the time. This is a rare occurrence, but it does happen, and then the diagnosis is almost impossible. When the friction sound is entirely absent

from the base of the heart over midsternum, and only audible towards the left of the cardiac area, I am inclined to regard it as purely pleuritic in character even though it persists during the cessation of breathing, but it is evident that though this is a probable view it is yet open to doubt. The subsequent progress of the case may show, in many cases, that the pleura is certainly affected, but that is no proof that the pericardium is not also implicated, or the pericardium may be assuredly diseased, and yet the friction sound may be wholly due to pleurisy. This is certainly one of the most difficult points in diagnosis, yet even in the most obscure case there cannot long be any great doubt whether the pleuritic or the pericardiac affection stands alone, or which is the essential disease when the two are combined; each case must, however, be decided on its own merits, and it is impossible to lay down any hard and fast lines applicable to all.

When effusion takes place into the pericardiac sac, the apex beat is said to be displaced upwards, the true apex beat is really obscured, and what we now feel is the part of the ventricle lying above it, and appearing to rise as more and more of the heart is separated from the anterior wall, with which the base of the heart always remains in contact. Hence, however large may be the effusion, basic friction, if it have once existed, is never effaced. Increased dulness from effusion is first of all to be detected at the base of the heart, but by-and-by the ordinary pyramidal dulness of the heart, base upwards, becomes reversed, and we have a pyramidal dulness with the base below, while the apex may rise as high as the clavicle or above it. The dulness may also extend beyond the apex beat to the left, especially if the patient lies upon his left side, but it is mobile, and on turning the patient on his right side, the dulness leaves the left and passes towards the right. Very great emphysema of the lungs may obscure this dulness, but cannot altogether annihilate it. In some cases friction is entirely absent throughout the whole course of the disease, not

merely in cases of simple effusion of serum, but even where abundance of fibrine is also effused. It is difficult always to account for this; some have supposed softness of the fibrine to be the cause, but the differences in regard to this cannot be sufficient to account for the entire absence of friction; feebleness of the cardiac action has most likely not a little to do with it, and possibly also some alteration in the physical condition of the parts, especially the lungs, overlying the heart, which may render them bad conductors of sound. If to absence of friction we have associated effusion into both pleuræ, then we are driven to surmise the pericarditis from general symptoms alone, the most important of which are a rapid, feeble, and dicrotic pulse, with feebleness or absence of cardiac impulse, and delirium, the latter being not infrequently, even in ordinary rheumatic pericarditis, the sole symptom which attracts attention, and it ought in these circumstances to direct that attention to the heart rather than the head. In January 1868 a case of this kind occurred in Ward VII., in the person of Andrew Dickson, a young man of 19, who had double pleurisy with considerable effusion into the left cavity, and a smaller effusion into the right; he had also occasional hæmoptysis, failure of the heart's impulse, rapid dicrotic pulse, and considerable delirium he had no rheumatism, and never any pain in the cardiac region. The pericardiac dulness was merged in the pleuritic dulness, and careful auscultation failed at any time to detect friction sound; the co-existence of pericarditis was surmised, but could not be detected. After death the pericarditis was found distended with reddish serum, and both its surfaces coated with shaggy, mammillated, blood-stained lymph; such cases are however unusual. The treatment of pericarditis almost invariably merges itself into the treatment of some other disease,—rheumatism, Bright's disease, tuberculosis, or pleurisy,—and I see no reason why, because the pericardium happens accidentally to become affected, we ought, therefore, to change our treatment, and at once "let loose the dogs of war." Where there is so much uncertainty, as in



therapeutics, we must be all the more cautious and wary in our treatment the more serious a disease is or has become, I see no reason to doubt that had the treatment of pericarditis formerly recommended been acted upon in my own cases, the results would have been widely different; it is not so long since that, terrified by the bug-bear of a name, many practitioners found themselves conscientiously driven to employ remedies uncalled for, either by the nature of the disease or the condition of the patient. Dr Stokes was one of the first to direct attention to the serious danger to the patient arising from thus raising a pathological hobgoblin by means of a name, and then running a muck at it through the vitals of the patient,\* and really there seems no reason why rheumatism, which neither requires mercury nor blood-letting, so long as it is confined to the joints of the limbs, should be supposed so urgently to require the use of those powerful, uncertain, and dangerous remedies, to the imminent risk of the patient, because it happens to attack that other joint which we call the pericardium. The mere detection of a little extra serum, or of a friction sound, is indeed no valid reason why we should jeopardise our patient by hazardous and unnecessary medication, and Bamberger,† Niemeyer,‡ Gairdner,§ and Bennet,|| have all shown that rheumatic pericarditis is a disease which will run its course more favourably, the less actively it is interfered with, and that all that is requisite is fitting constitutional treatment and local palliatives, and that the constitutional treatment must be directed to the disease with which the pericarditis is associated, and not to pericarditis itself. We are now-a-days even more able to carry this out than formerly, because while Bamberger and Niemeyer recommend leeching for relief of the pain, we can relieve this more effectually and more rapidly than by leeching, and at less risk to the patient, by the subcutaneous

\* *The Diseases of the Heart and Aorta*, Dublin, 1854, p. 82.

† *Lehrbuch der Krankheiten des Herzens*, Wien, 1857, pp. 131-133, &c.

‡ *A Text-Book of Practical Medicine*, London, 1871, p. 389.

§ *Ed. Med. Journal*, Jan. 1861, p. 632.

|| *The Principles and Practice of Medicine*, Edinburgh, 1865, 4th ed., p. 575.

injection of morphia, and by subsequently, if necessary, keeping up the narcotism by means of small repeated doses of chloral, which is not more useful as a sedative than as an antiphlogistic. A light, warm poultice should be kept constantly over the cardiac region, and that constitutional treatment employed which seems most suitable to the patient's condition. Thus, if the pulse be rapid or feeble, digitalis should be resorted to combined with an alkali, such as potass or ammonia; but blisters irritate the patient always more or less, and should be shunned, as they tend to excite the heart's action, which it is our object to keep quiet. In fact, we treat the pericarditis simply as a part of the general rheumatic attack, requiring a little more than the usual attention in the way of warmth, and relieving pain.

In occasional instances it may become a question as to whether the pericardium should be tapped or not. It is our duty to obviate death from any preventible cause, and therefore, if the heart's action seem oppressed and life endangered by the amount of effusion, we ought certainly to tap the pericardium; but in all ordinary cases this is quite uncalled for, and when it is indicated, that indication is only too frequently a sign that the powers of the constitution are in so depressed a condition as to render the relief thus obtained merely temporary. In the following case this question arose, but as the constitutional depression was much more serious than appeared to be consistent with the amount of effusion present, it was postponed from a feeling that there was something unusual in the case not to be eliminated by tapping, till almost at the last moment my resident, Mr Saundby,\* did operate, but with only temporary relief, and the nature of this case was such that it could not have been otherwise.

CASE XXVIII.—Thomas Fraser, aged 13, was admitted into Ward V., bed 1, on 21st of May 1874, complaining of cough with pain and tightness of the chest. His history was that

\* Mr Saundby has published this case, with remarks, in the *Ed. Med. Jour.* March 1875, p. 799.



about eight days before his admission he had gone to bed apparently quite well, and in the morning found himself unable to get up from severe illness, presenting the symptoms just described. He was a poor ill-thriven boy, whose father had apparently died of phthisis, at least of some disease accompanied by cough and emaciation, his mother was still alive, but sickly. The patient lay easily on his right side; his face was puffy, his insteps slightly cedematous, his countenance expressed both anxiety and pain. His temperature on admission was  $102^{\circ}4$ , and during the whole of his illness it varied between  $102^{\circ}$  and  $99^{\circ}$ , being usually a degree higher at night than in the morning; on the day of his death his temperature was  $98^{\circ}$ , the previous day it was  $100^{\circ}$ . His pulse was 80, soft and very irregular in character, but especially in force, and he complained of great dyspnoea and tightness across the chest. On inspection the precordia seemed to bulge more than usual. Upon palpation no cardiac impulse could be felt at all. On percussion one inch from the left of the sternum, the dulness did not rise above the third rib, but from that rib passed downwards till it merged in the stomach resonance; at the level of the fourth rib, the transverse dulness commenced two inches to the right of the sternum, and extended across the sternum to a distance of five more—seven inches in all. Upon auscultation the heart's sounds were to be heard normal enough in all the areas when the patient held his breath. Over the precordial region a loud friction sound was audible with the respiration, ceasing when the patient held his breath. His respirations numbered thirty-six, he had great pain in coughing expectoration mucous and very trifling. Anteriorly over the lungs the percussion note was normal, except when interfered with by the distended pericardium. Posteriorly there was dulness over the left base. On auscultation there were friction sounds audible over the left base anteriorly and posteriorly, and some rhonchi over both sides. His tongue was slightly coated, appetite defective, bowels loose. The quantity of the urine could not be estimated, as much of it passed with his stools;



what could be collected was of a reddish colour, acid, spec. grav. 1027, and contained no sugar, albumen, or bile, the chlorides were present, and it deposited urates. His face and feet gradually became more and more œdematous, but there was no anasarca to speak of. Permission was given to tap the pericardium on the symptoms becoming urgent, without much hope of relief, but from sheer unwillingness to leave any stone unturned, and on the evening of 10th June, the pulse at the wrist having become imperceptible, he was tapped and thirty ounces of pus drawn off, his pulse returned, was 84 in a minute, and for a time he rallied, but died at 1.30 on the morning of the 11th June. At the autopsy on 12th June some serous fluid was found in the left pleura, and there were recent adhesions over the lower part of the left pleura, chiefly anteriorly; there were 36 ounces of pus in the pericardium; the heart was firmly adherent to the posterior part of the pericardium, and covered anteriorly with purulent lymph. At the base of the right lung there was an abscess close to the pericardium and adherent to it, but no communication could be traced. The rest of the autopsy was unimportant. In spite of the absence of communication, it seemed a very probable matter that in this case the purulent pericarditis was due to rupture of the abscess in the right lung into the pericardium, the subsequent pericarditis closing up the opening, and the left pleurisy arising from contiguity in a feeble and exhausted constitution. But little relief could be expected from tapping in such a case, but it can be so easily done now-a-days by means of one or other of the aspirateurs that it seemed a right thing to try to prolong life by its means. The effusion was never very great, and the constitutional depression was so excessive in comparison with its amount, that it could only be permitted as a *dernier ressort*, and possibly would have been as well left alone; but we never know what may happen, and it is always right to prevent death if possible, on the chance of some favourable change taking place. Even in purulent pericarditis this is not impossible, for

the elements of pus are more or less present in every pericarditis, and pus may be only a transitional stage, and may result in the breaking down of the cell elements, the formation of a pathological cream, its complete absorption, and the perfect cure of the disease, though of course this can only rarely be the case.

The termination of pericarditis in complete adhesion of the pericardium to the heart is of great pathological importance, but of little practical interest, because it is almost impossible to diagnose; we may with greater or less certainty surmise it, but unless in very exceptional cases\* we can never be certain of it. Of course its occurrence is never without an important influence on the future of the patient, but the degree of importance depends very largely upon the extent to which the integrity and nutrition of the cardiac muscle are involved. These results, however, it is impossible to diagnose or to predict. One of the most extraordinary instances of this mode of termination of pericarditis is to be found in the University Museum here, and is referred to by Dr Burns in his "Observations on Diseases of the Heart," as one in which the pericardium was unusually adherent, and the ventricles so ossified (they are really atheromatous) that, except for a cubic inch over the apex, they were as "firm as the skull," yet the patient had never had any palpitation nor pain in the region of the heart.† Fibrinous effusion into the pericardium may compress the coronary arteries and lead to angina and cardiac dilatation, and an adherent pericardium may even be associated with hypertrophy, though this must be rare, but it is impossible to predict these alterations, and almost impossible to surmise them, even with the history of the case before us.

It has twice occurred to me to find a loud friction sound over the whole cardiac area without any objective symptoms whatever. The patients certainly felt themselves not quite right, but there was no rise of temperature, nor any pain; neither was any other disease present which might account for

\* *Vide* Lecture I. p. 2.

† *Op. cit.* p. 131.

a latent pericarditis, in particular there was no kidney disease. In one of the cases the friction sound came and went in the most unaccountable manner, while the variations of intensity in the other case,\* though evident enough, were not so remarkable. It is alleged, I believe, by some anatomists that muscular fibres are found in the pericardium, and the phenomena presented by these two cases seemed more easily explicable on the supposition that the heart was spasmodically grasped by the pericardium than on any other theory whatever.

Acute endocarditis is largely a pathological disease, and not a clinical one. The systolic apex murmur supposed by so many to indicate endocarditis, when it occurs in acute rheumatism, in much the larger proportion of cases depends solely upon dilatation of a perfectly curable character,† while any symptoms peculiar to this affection, when it does exist, are merged in those of the concomitant pericarditis, or simply in those of the general disease. I have thrice had the opportunity of watching the gradual development of mitral stenosis from a state of perfect health in non-rheumatic cases, but the symptoms exhibited were not distinctive enough to enable me to lay down any rules for their recognition, in the early stages at least when this recognition would be of most importance. Indeed, apart from those general diseases,—Rheumatism, Chorea, Pyæmia, and Bright's disease,—in which the form of heart affection is only too often correctly surmised when cardiac symptoms of any severity are present, the general history of endocarditis, in its subacute or chronic forms is most closely allied with endarteritis, with which it is often associated, and its diagnosis can only be properly considered in connection with this disease, exophthalmic goitre in its early stage, and one form of the neurotic heart, in all of which alterations in the cardiac

\* Robert Hume, admitted to Ward XXXVI., New Royal Infirmary, June 23, and discharged July 13, 1880, the friction still occasionally recurring. I see this man occasionally going about the picture of health, but have not lately examined him.

† *Vide* Lecture VI., p. 185 *passim*.



rhythm and variations in the intra-arterial blood pressure form the most interesting and predominant symptoms. Myocarditis belongs to the same category. I shall not therefore enter upon the clinical history of these affections at present, but content myself with giving an account of one or two cases in which more or less extensive myocarditis was found after death, when nothing of the kind had been suspected beforehand. The following are the most interesting cases of this kind which have occurred to me :—

CASE XXIX.—Francis Lynch, admitted first of all on 23d June 1868, discharged improved on 11th July; readmitted on 5th October, discharged improved on 17th December; readmitted on 2d January, and died in Ward VII. on 6th May 1869. This patient had a mitral bruit of a shifting character, it disappeared for weeks at a time altogether, at other times it was loud and distinct, the tricuspid bruit was equally variable; it was impossible to make anything of his cardiac symptoms, except that there was a gradually increasing failure of the cardiac power accompanied by an occasional murmur, now more distinctly mitral, and at other times more distinctly tricuspid; he was repeatedly seen by my colleagues, but nothing more definite was elicited. At the autopsy on 6th May 1869, the pericardium contained a considerable quantity of yellowish serum, the heart was enlarged, the apex rounded, the right ventricle projecting a little beyond the left. The aortic and pulmonary valves were normal. The circumference of the pulmonary artery measured three and a quarter inches. The tricuspid orifice measured six and three-quarter inches. The aortic orifice measured three inches, and the mitral orifice five inches in circumference. The right ventricle was dilated and distinctly hypertrophied. The left auricle was somewhat dilated. The left ventricle was in some parts atrophic, but the muscoli papillares were distinctly hypertrophied. A considerable part of the ventricular wall was atrophied and reduced to one-half its natural thickness; over this part the endocardium seemed to be continuous. There was no aneurismal dilatation

of the wall, but the cardiac cavity was increased by half the thickness of the ventricle. Adherent to the atrophied part there was a buff coloured clot, firm on its surface, and partially laminated, the superficial layers being redder and apparently more recent; on closer examination the degeneration was found to be confined to the inner half of the ventricular wall, the other half being composed of apparently natural muscular fibre. Quite at the apex, and at the lower part of the septum, the atrophied portion was greater than that remaining, but higher up the muscular substance gradually increased till it occupied the whole thickness of the cardiac wall. The fibrous part contained some dense white lines, and was otherwise composed of fine fibrous tissue. Microscopically examined, the muscular fibre in every part of the heart was found to be fatty. In the atrophic portion there were found traces of occluded blood-vessels, with granules and crystals of blood pigment contained within them, and mingled with them there was a large amount of dense fibrous tissue, with little patches of fat and traces of muscular fibre. The branches of the coronary artery leading to the atrophied parts were occluded. The left pleural cavity contained a considerable amount of serum which compressed the lower lobe of the corresponding lung. The right lung was congested and œdematous. There was lateral curvature of the spine in the dorsal region. The rest of the body was not examined. This patient never had rheumatic fever, and was perfectly healthy up to January 1868, when he caught cold, shortly after developed shortness of breath, and ever after continued ailing till he died. I do not know of any other case of myocarditis so long under observation and seen by so many physicians, and yet his precise condition was not even surmised.

Another most interesting case is represented solely by the annexed drawings, which exhibit the microscopic appearances of his heart. You will see that the connective tissue of the pericardium, the sub-pericardial fat, and the muscular fibres, are all infiltrated with a hyperplasia of the corpuscular



element. The patient to whom this heart belonged laboured under double aortic disease, but had improved greatly under treatment. He had a good firm apex beat, considered himself quite well, had got his discharge, and was just about to leave when he died suddenly from asystole. On dissection his heart was flabby, of a yellowish tint, and was pronounced fatty. My resident, Dr C. S. Roy, secured the heart, and, on examining it microscopically, discovered the lesion described. The drawings, figs. 20 and 21, were made by Dr Roy from specimens prepared by himself.

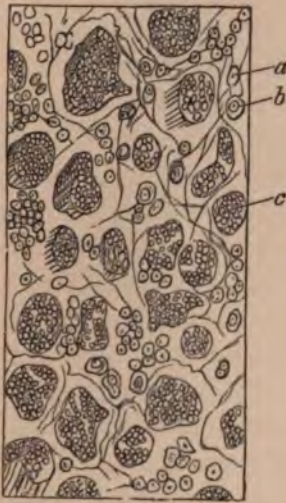


Fig. 21.

Fig. 21.—Section of muscular tissue of heart. *a*, Nucleus; *b*, nucleated cell; *c*, transverse section of muscular fibre.



Fig. 22.

Fig. 22.—Vertical section of pericardium and sub-pericardial connective tissue. *a*, Pericardial fibrous tissue infiltrated with nuclei; *b*, fat cell; *c*, nucleus; *d*, capillary vessel transversely divided.

I have no idea to what this condition would ultimately have led had the patient been spared, whether to suppuration, or to fibrosis of the heart with destruction of the muscular tissue.

But the most remarkable heart of this kind I have ever seen, was that of Case XXX., Ralph Scott, admitted into Ward V. on June 22, 1877. This man was forty five-years of age,



and of very intemperate habits. He was not known to have ever suffered from rheumatic fever, but had for long suffered from cough and shortness of breath, and he was admitted into the Infirmary on account of the severity of these symptoms. He was found to have considerable general dropsy with cedematous lungs, a very large heart, and both a systolic mitral and a tricuspid bruit, but nothing peculiar was noted in regard to the action of the heart. Four days after admission, on June 26, 1877, he died suddenly after an epileptiform convulsion. Autopsy, June 27, 1877. The body was that of a man of rather a large frame, with a good deal of anasarca, especially in the lower extremities. Thorax: The pericardium was firmly attached over the whole surface of the heart by dense old adhesions. The heart itself was enormously enlarged, weighing two pounds; all of its chambers were much dilated, the dilatation of the right ventricle being extreme. The cavity of the left ventricle was greatly dilated, and its wall much hypertrophied, measuring from three quarters of an inch to one inch in thickness. In the upper half of this ventricle the muscular tissue was sound, but about the apex the muscular fibre was largely replaced by cicatricial tissue so dense as to creak under the knife. About one inch and a half above the apex there was a pouch-like bulging of the ventricular wall, which would have developed into an aneurism had the patient lived. Over the whole area of fibrous induration the pericardial adhesions were specially dense, and the endocardium was thick and of an opaque yellow colour. The aortic orifice was normal in size, measuring three inches in circumference. The aortic valves were competent and natural. The mitral orifice was dilated, admitting five fingers (cone diameter, 1.75 inch). The anterior cusp of the valve was natural, but the posterior cusp was in a state of advanced disease, its left half was natural but the right half was greatly thickened, and was so curved and bulged out from behind, by the pressure of the blood upon its posterior or ventricular surface, as to press forwards as a rounded or rigid sac against the surface of the anterior cusp. Behind the

concave surface of the diseased cusp, the posterior wall of the ventricle was hollowed out into a concavity, and presented the same thickening of the endocardium and fibrous induration of the muscular tissue, as was noted in regard to the ventricular wall at and near the apex. Owing to the differentiation of the posterior cusp into a sound and a diseased portion, the whole arrangement looked curiously like a tricuspid rather than a bicuspid valve. The right auriculo-ventricular opening was enlarged, admitting five fingers (cone diameter, 1.75 inch). The aorta was normal in size but very atheromatous, and some of its intercostal branches were partially occluded by the consequent thickening of the "tunica intima," but the coronary arteries were not obstructed.\* I omit the remainder of the autopsy as unimportant. The fact that a heart so crippled as this was yet able to discharge its function fairly well, for what no doubt—from its size, and the fact that its history was entirely lost—must have been many years, ought to encourage us in our attempts to remedy even cases which seem to be apparently hopeless.

I have mentioned that cardiac hypertrophy may occur as a result of pericarditis apart from valvular disease. That it does so occur there can be no doubt, but almost all the cases of excited action with hypertrophy and without valvular disease that have occurred to me, have either had a distinct syphilitic history or have yielded to anti-syphilitic treatment, the solution of the red iodide of mercury or large doses of iodide of potassium. In one such case, however, which was long under the treatment of various physicians before he came under my care, the disease was, according to report, much ameliorated by spare diet in its earlier stages. There was in him no history of pericarditis, and no distinct history of syphilis; latterly, the changes incident to advancing age permitted the use of a more liberal diet and even of stimulants in small doses with advantage, arsenic in small doses also proved greatly beneficial, but in all these cases digitalis is to be avoided and is injurious.

\* Condensed from the Pathological Reports of the Royal Infirmary.



## LECTURE XIV.

ON THE ACTION OF DIGITALIS ON THE HEART, WITH A FEW  
REMARKS UPON THE THERAPEUTICS OF CARDIAC DISEASE  
GENERALLY.

GENTLEMEN,—In speaking of the various forms of cardiac disease, I have already alluded, somewhat cursorily, to the best methods of treating them. At present I wish to go a little more fully into the mode of action of one of the three or four remedies we chiefly employ, while I shall also make what supplementary remarks may seem necessary upon the others, so that you may not only understand the reasons for which we employ these agents in any special case, but may also so fully comprehend their action, as we at present know it, that you may be able to extend or modify their use in those cases which may come under your own special observation.

In the treatment of disease of the heart there is but one remedy, *par excellence*—DIGITALIS, the action of which it is most important thoroughly to understand, because the use we make of it depends entirely upon the conception we have as to its mode of action; for if we regard it, as some even yet do, as weakening and depressing the heart's action, it is obvious that its use will be much more restricted, and confined to quite a different class of cases, than if we look upon it as a tonic and stimulant of the heart. I need not tell you in which light I regard it, as you have often enough had occasion to observe the almost marvellous results which follow its bold yet



judicious employment as a cardiac stimulant, even in cases of aortic regurgitation, cases which are only too generally excluded from the benefits of this remedy by many who hold otherwise sound views as to the action of the drug.\* Foxglove—or more properly Folk's-glove—the Fairies' glove—is mentioned in Saxon writings of the eleventh century; but it was so little known to the learned that it had no Latin name till the beginning of the sixteenth century (1535), when Fuchsius, professor of medicine in Tübingen, gave it the name of *Digitalis*†—a translation of its German name, Finger-but—which it still retains. In 1597 old Gerarde recounts its various uses, and Parkinson, the herbalist to Charles I., regrets that few physicians used it; showing, too, how wonderfully full his practical knowledge of its actions was by claiming it as an important remedy for epilepsy.‡ It appears among the list of simples in the London Pharmacopœia in 1650, but it seems to have been chiefly employed as an external application in scrofulous ulcers and glandular enlargements. Neglected by the profession, it was nevertheless in great repute among the common folk in certain districts of England as a cure for dropsy; and at length, in the end of last century (1775), Withering rescued it from its undeserved obscurity, and laid down correctly enough the main indications for its use as a diuretic. “It seldom,” he says, “succeeds in men of great natural strength, of firm fibre, of warm skin, of florid complexion, or in those of tight and cordy pulse;” but he adds, “on the contrary, if the pulse be feeble and intermitting, the countenance pale, the lips livid, the skin cold, the swollen belly soft and fluctuating, or the anasaruous limbs readily pitting under the pressure of the finger, we may expect the diuretic effects to follow in a kindly manner.”§ Here you will see that we have no indication of any sedative action, but on the contrary a distinct statement that it acted best in

\* Even by Dr Fothergill, *vide op. cit.* p. 52.

† *Vide* Lauder Brunton *On Digitalis*. London, 1868, p. 3.

‡ Which it is, in cardiac epilepsy, *vide antea* p. 258.

§ *An Account of the Foxglove, &c.*, Birmingham, 1785, p. 189.

conditions of depressed vitality, and was therefore a stimulant of some sort. Yet Withering knew that in large doses it depressed the heart's action and produced nausea and vomiting, but he also knew that this sedative action was fatal to the development of its diuretic properties, and he points out that in this it differs decidedly from squill, which never acts so powerfully as a diuretic than when it produces a slight degree of nausea.\* This antagonism between the sedative and diuretic properties of digitalis is also pointed out by Christison in his "Dispensatory," and is really the key to the safe and successful employment of the drug; though it is remarkable how long it was before this sedative action was recognised to be merely the result of the excessive development of the stimulant action of the remedy, a fact which is perhaps even yet not so fully acknowledged as it ought to be, and must be, before we can have that confidence in the drug necessary for the production of its most brilliant therapeutic results. Shortly after the publication of Withering's work, digitalis was received into the London Pharmacopœia (1809), not merely mentioned among the simples, but the preparation of the Infusion and Tincture both described; and it has ever since continued to be one of our most valued therapeutic agents, though it is only in quite recent times that experimental physiology has so thoroughly determined its mode of action as greatly to extend its practical application and strengthen our confidence in its employment.

We know very well that in weak hearts, and in all hearts affected with valvular disease of whatever nature, in which compensation is ruptured or imperfect, the tendency is to death from asystole, as it is termed, that is, to death with the heart in diastole from failure of its contractile power. In this condition, in its best-marked and most striking form, the heart empties itself imperfectly, each contraction of the ventricle only expelling a small quantity of blood from its upper part, often barely sufficient to maintain temporarily a life

\* *Op. cit.* p. 3.

which is gradually being lost through diminution of the blood-pressure in the arterial system, with all the direful effects already described as flowing from this, one of the earliest of these being the diminished secretion of urine.\*

Now this is very much the condition of heart produced by aconite poisoning. It is important to remember this, because aconite and digitalis have both been regarded as cardiac sedatives, and have been employed by some indiscriminately to the great detriment of their patients. But if we give a frog aconite till its heart is brought to a standstill in diastole, and then administer digitalis, the distended ventricle gradually and slowly recovers itself, and by and by returns to its normal condition as to contraction and distention.† This important experiment not only teaches us the action of digitalis, but is also well fitted to give us such confidence in its powers as will enable us to use it with safety and success in many cases of disease in which we would not otherwise have ventured to employ it. And a continuance of the experiment is not only fitted to confirm our confidence in the stimulating action of digitalis, but also to explain the consequences of a poisonous dose, and to show how a most powerful and, it may be, fatal sedative effect may result from the over-action of a tonic drug. For if we continue to administer digitalis freely to the frog already described, or if we employ it with equal freedom in a healthy frog, the cardiac systole becomes gradually longer and more persistent, and the diastolic dilatation less complete, especially at the apex, which remains white and firmly contracted, failing to relax during the diastole. This abnormal condition gradually extends upwards over the whole of the ventricle, the diastolic dilatation becoming less and less complete, till the heart comes to a standstill in firm contraction, just the reverse of its state in aconite poisoning. Similar results are obtained both in birds and mammals.‡ Now you

\* *Vide antea*, p. 277.

† *Vide Fothergill On Digitalis*, p. 6.

‡ *Fothergill, op. cit.* p. 5.



see how the over-action of digitalis may produce dangerous results, similar in character in their effects upon the circulation to those of aconite poisoning, though from a different cause. In aconite poisoning or in threatened asystole the aortic pressure falls because the over-distended ventricle is unable to contract upon its contents, each contraction only succeeding in expelling a small quantity of blood off the top of the distended ventricle; in digitalis poisoning, on the other hand, the aortic pressure falls because the over-contracted ventricle permits but little blood to get into it and can consequently send but little forward. The result in both instances is similar, but the cause in the one case is just the reverse of that in the other. It is of importance to remember this as a key to the manner in which digitalis acts,—viz., that while it slows the heart's action it also increases the strength of the ventricular systole, and diminishes the amount of diastolic dilatation, this dilatation lessening *pari passu* with the increased action of the drug.

Digitalis may thus be made to act pretty much as we wish, and by appropriate dosage a patient may be enabled to take it continuously for years with nothing but increasing benefit, or he may be compelled to give it up, after but a few doses, from symptoms of incipient poisoning. We have the matter entirely in our own hands. The cumulative action ascribed to digitalis is neither mythical, nor mysterious, it is simply a result depending on the relation between absorption and elimination common to it and to many other drugs. If we remember this, and how digitalis acts, we shall find it one of the most manageable and most certain of remedies. If, for instance, we wish simply to improve the nutrition of the heart, we must give only a tonic dose, and give it so that it has only a tonic action; that is to say we must give a dose just large enough slightly to increase the force of the cardiac systole without appreciably slowing the rate of contraction, and we must repeat this at such an interval as will secure that its *action* has passed entirely off, though its *effect* still persists.

The *action* of such a dose is the slight augmentation of the force of the cardiac systole, with the trifling but not unimportant increase in the blood tension within the coronary arteries resulting from it; while the *effect* is the improved nutrition of the muscular fibres and ganglia of the heart, which it is our object still further to promote by a renewal of the dose neither too quickly repeated nor too long delayed. By this mode of administration we secure a gradual improvement in the nutrition of the cardiac muscle, as well as an increase in the reserve power of the ganglia, so that by slow and imperceptible degrees a weak heart is made a strong one, every function is proportionately benefited, and a new vigour permeates the frame.

If however we wish to act more rapidly and decidedly on the heart and circulation, we must—whatever dose be given—repeat it at short intervals, so that *the action* of each dose may be reinforced by that following it, until the desired result is attained. This is the mode in which we employ digitalis when we wish to contract the heart, or to remove dropsy. In cardiac dilatation it is most successful when the affection is recent, and it becomes less and less useful the larger and more hypertrophic the heart gets, till at last it fails utterly in the later stages of fibro-fatty degeneration. It also occasionally fails when the cardiac nutrition is shackled by a completely or very extensively adherent pericardium.

In selecting the dose for any special case idiosyncrasy counts for something, as some are undoubtedly more sensitive to the action of digitalis than others. But a careful watching of the first few doses will settle this matter, and we can then readily discount it if present. Bulk of body, but especially a greater or less degree of plethora, are also not to be disregarded in selecting the dose, as feeble and anæmic persons are undoubtedly more rapidly saturated than those possessed of a greater blood mass. Further, inflammatory conditions have long been held to inhibit more or less completely the action of this drug, and there seems to be some truth in this.



The powder of digitalis, prepared from the carefully dried leaves, was looked upon by Withering as its most active preparation; it is still a useful one, where a pill is preferred, but it is apt to irritate the stomach, and is not now regarded as superior in any way to a well prepared infusion or tincture. Withering supposed that thirty grains of the powdered leaves in bulk were about equal to forty grains in infusion, and that this quantity could generally be taken before nausea occurred. Blackall\* pointed out that there was but little difference between the powder and the infusion, and the action of any preparation may thus be reasonably calculated by the number of grains of powdered leaves it represents. It is well to remember this; the superior diuretic virtues ascribed by many to the infusion of the Edinburgh pharmacopœia, are readily understood when we reflect that by its use the equivalent of from three to twelve grains of the leaves is given thrice a day or oftener. But half an ounce of the British pharmacopœia infusion contains only one grain and a half of the leaves, and ten minims of the B. P. tincture little more than one grain. One ounce of our present infusion, and half a drachm of our present tincture represent therefore the minimum dose of the former infusion, and in these doses either preparation will be found to emulate its virtues. For ordinary use the tincture is the most uniform and reliable preparation, but a fresh infusion carefully prepared from leaves not over one year old is at least as good. Nativelles granules, each containing  $\frac{1}{4}$  of a milligramme, or about  $\frac{1}{280}$  of a grain of crystallized digitaline, are also perfectly reliable and are each equivalent to rather more than one grain of the crude drug. They seem to be less liable to excite nausea than other preparations, and are invaluable for hypodermic injection, in those rare cases in which this method of ingestion is our sole resource.

One grain of the crude drug, or its equivalent in one or other preparation, given every twelve hours, is a purely tonic dose in all but the most exceptional cases. In these excep-

\* *Observations on the Nature and Cure of Dropsy.* London, 1814, p. 312.



tional cases even this dose, and in all others any larger dose is cumulative in its action, so that after a longer or shorter time certain sensible results are found to follow. These are a primary increase in the flow of urine, with subsequent diminution; nausea and vomiting; or a preternatural slowing of the pulse. Purging is reckoned by Withering one of the actions of digitalis; it occasionally occurs, but whether *propter* or merely *post* I have never been able to satisfy myself; when it does happen diuresis is always lessened but not necessarily put a stop to. When we wish to remove dropsy, or contract a dilated heart, the digitalis should be given at much shorter intervals, and the more urgent the case the shorter should be the interval and the larger the dose. The equivalent of rather more than forty grains may usually be given before symptoms of saturation appear, and the larger the dose and the more rapidly it has been ingested, so much the more decided these symptoms are. But nothing but good will follow provided we stop the moment the urine falls, the pulse slows, or nausea occurs. When saturation is produced more slowly by the ingestion of a smaller dose at longer intervals and continued for a long time, the primary slight diminution of the urine, or not very marked slowing of the heart's action, may escape observation unless carefully watched for. In such cases the heart sometimes becomes so contracted that its sounds are reduced to a toneless tic-tac, while at others the slightly thumping pulse of saturation thus produced quickly passes into the *pulsus bigeminus*, or into the allorhythmic pulse of digitalis poisoning, conditions not dangerous in themselves nor even attended by much anxiety provided the drug is at once stopped, and the recumbent posture enjoined. When giving digitalis for only tonic purposes we must carefully watch, for a week or two, for any signs of saturation, before finally fixing on the dose, which should not as a rule exceed one grain in twelve hours. For all other purposes I am satisfied it is better to give full doses for a short time rather than to give smaller doses for a longer period. Some-

fatty heart,\* and we all know very well that by far the larger proportion of hearts having a feeble impulse are only weak and dilated and not fatty; we also know that such hearts peremptorily require the somewhat free use of digitalis, and are much benefited by it, and we must not be driven from its employment by any mere theoretical hypothesis. Of course, rupture of the degenerated muscular fibres may occur at any moment whether digitalis be employed or not, a string of oil globules has but little cohesion at any time: but the following case affords pretty conclusive proof that digitalis is in itself unlikely to be an active agent in producing this rupture, while it proves as conclusively the impossibility of diagnosing fatty degeneration of the heart:—

CASE XXXIII.—John Steven, a sailor, aged 64, admitted to Ward V. (from the clinical wards) on the 24th July 1875, complaining of cough, shortness of breath, palpitation, brawny swelling of the legs, and some ascites. He admitted having been a hard drinker. The cardiac action was rapid, irregular, and intermittent, the apex beat was quite distinct though not forcible, and was felt outside of and below its normal position. The sounds at the base of the heart were very much obscured by bronchitic rhonchi, but no distinct murmur could be distinguished. The mitral sound was impure but without murmur; a murmur of tricuspid regurgitation was, however, occasionally audible. The patient's liver was not enlarged, but he had a trace of albumen in his urine, which three weeks before his death became considerably increased. For a week before death the sputa became rusty. This patient was regarded as labouring under a weak, dilated, and probably a fatty heart, the latter part of the diagnosis being based not upon any physical sign or symptom, but mainly on his previous drunken habits. During the ten weeks he was under treatment in Ward V. the only relief he obtained was from large doses of digitalis; fifteen minims of the tincture every four hours was his average dose, but sometimes he got more, never less. I did not see him for

\* *Vide antea*, p. 309.



two weeks before his death, and at that time there seemed still a prospect of the digitalis restoring his cardiac power; he died, however, apparently from asthenia, culminating in asystole, upon October 5, 1875. On dissection, on October 7, the body was found to be greatly anasarcaous and considerably decomposed. The heart was found to weigh twenty-four ounces, the parietal and visceral layers of the pericardium were adherent throughout. The connecting lymph was recent except posteriorly, where it was rather fibrous. The heart was both dilated and hypertrophied; all its valves were thickened by atheroma but competent, except the aortic valve which was slightly incompetent. The cavities of the heart were filled with blood, which was black and fluid. The cardiac muscle was everywhere soft, friable and could easily be penetrated by the finger; upon microscopic examination it was found to be in an advanced state of fatty degeneration, the striation being entirely lost. The lungs were firmly adherent throughout their whole extent, intensely congested, and of a dark colour; the right weighed 5 lb. 3 oz., the left 2 lb. 4 oz. The liver was fatty, congested, and weighed 3 lb. 13 oz. The spleen was congested and its capsule thickened; it weighed 5 $\frac{3}{4}$  oz. The kidneys were in a state of advanced fatty degeneration, as proved by microscopic examination; the right weighed 8 oz., the left 7 oz.\* To those who witnessed the very great utility of digitalis during the life of this patient, and who afterwards saw the extremely fatty state of his heart, nothing could prove more instructive as to the power of the drug so long as a trace of muscular fibre remains, or as to the little danger fatty hearts are exposed to from the action of digitalis. In fact, even in aged people, with a feeble impulse and tendency to syncope, in whom fatty degeneration may reasonably enough be suspected, I have never seen any reason to withhold digitalis, and have always hitherto been rewarded by considerable and well-marked improvement. In the case of one gentleman, now eighty-eight years of age, who came under my

\* Extracted from the Pathological Records of the Royal Infirmary.



care some ten years ago with the symptoms mentioned, the continuous administration of digitalis for several years resulted in the disappearance of all tendency to syncope, and in the reproduction of a firm and vigorous cardiac impulse. Granting, then, that there may be a grain of truth in Dr Brunton's theoretical objection, and I am not sure that there is even so much, still in the face of all the difficulties surrounding the diagnosis of a fatty from a merely feeble heart, I think that for our patients' sake we are quite justified in treating all such cases as merely weak hearts.

We have no remedy that can replace digitalis in the treatment of cardiac disease. Ergot and belladonna both act upon unstriated muscular fibre, and might be supposed to be useful, the latter especially having been recommended by Dr John Harley; but though it certainly increases the force of the heart's action it is also apt to increase its frequency, and thus to diminish instead of increasing the cardiac rest.\* It can never, therefore, replace digitalis, though occasionally I have thought it useful in maintaining the force of the heart's action in cases of aortic incompetence where digitalis had already been freely given and in which a pause in its use seemed desirable; ergot I have never found of the slightest use. The dajasck, or arrow-poison of Borneo, is said to kill with the heart in systole, and may possibly become a valuable addition to our therapeutic agents; at present it is merely a scientific curiosity.

STRYCHNIA is well known as a powerful nervine tonic, but it is perhaps scarcely so well known as it deserves to be as a stimulant to the cardiac ganglia.† Where the heart is more irritable than weak, that is where dilatation has not obviously commenced, and yet the want of tone in the cardiac walls, and ganglia, is sufficiently unpleasantly revealed

\* Harley himself points this out; he says, "After moderate doses the whole circulation is increased in force and rapidity,"—*The Old Vegetable Neurotics*. London 1869, p. 221.

† For an interesting explanation of the action of strychnia on the heart *vide* a paper by Drs Lauder Brunton and Cash at p. 229 of vol. xvi. of *St Bartholomew's Hospital Reports*.

by all those allorhythmic vagaries which the most trifling reflex irritation is prone to occasion in such cases, then strychnia is indicated much more than digitalis. Whenever, in short, the cardiac energy is defective without any evident structural lesion, strychnia continued for a considerable time will be found to induce a most marked improvement. It matters not how the want of energy is revealed, by intermission, irregularity, or rapid action, the long-continued use of strychnia puts all to rights in most cases in what seems to be an almost marvellous manner. A few weeks often work wonders in such cases, in most, however, we must wait for months before we reap the full benefit. Even when there is actual structural lesion strychnia is still most useful, and in these cases it is best to give this tonic either alone, or in combination with arsenic and iron along with food, and tonic doses of digitalis night and morning. Of course we must also endeavour to remove all probable sources of irritation from these allorhythmic hearts, as by washing out with Vichy water in the case of acid secretions or giving compound galbanum pills when flatulence is present, besides regulating the diet appropriately.

Next to digitalis ARSENIC is probably our most important agent in the treatment of cardiac disease; its neurotic action is undoubtedly its most remarkable one, and its effect in removing cardiac pain of an anginous character is really something marvellous. I need not, however, enter further upon this subject here, as I have already alluded somewhat fully to this action, as well as to the important tonic power which it seems to exert upon the cardiac muscle,\* in virtue of which it may be employed not only as an adjuvant to digitalis, but as a cardiac tonic in certain cases where that drug is inadmissible as in cases of hypertrophy when the muscular power is beginning to fail, but is yet too great and too easily excited to permit the use of digitalis. I may also add that arsenic almost invariably regulates the bowels, and seems in this way to unload the liver without any risk of undue purgation, this being not in-

\* *Vide antea*, pp. 108 and 314.

frequently a matter of some consequence in feeble and dilated hearts, and productive of no inconsiderable comfort to the patient. The relief to cardiac pain and the regulative action on the bowels resulting from the use of arsenic, in small doses, renders it a most important adjunct to digitalis in the treatment of cardiac disease; and this combination will be found to be one most generally useful, the utility of which may occasionally be supplemented by the use of IRON in one or other of its many preparations. But iron, especially in the form of the perchloride, frequently requires much caution in its use from its disturbing effect on the stomach, and the cardiac irritation which so surely results from that. Five minim doses of the tincture of the perchloride of iron, twice a day, are about as much as we frequently dare give in such cases, and even this minute dose will not infrequently be found to disagree and require to be given up. The protochloride is a much more easily assimilated preparation than the perchloride, and, either in the form of dragées or syrup, it is a very pleasant and efficacious mode of administering iron. Other preparations, such as the citrate of iron and ammonia, the citrate of quinine and iron, the phosphate and hypophosphite of iron, &c., may also be given freely, but when iron in large doses is peremptorily required, as in chlorosis, then it seems to be better borne as Bland's pills \* than in any other form.

I have already told you that the subcutaneous injection of MORPHIA is a most important agent in enabling us to give immediate relief to cardiac pain, and so to soothe the patient as to secure his necessary rest,† a matter of no less importance for his relief than for his comfort. I have also already entered somewhat fully into the uses of NITRITE OF AMYL and of CHLOROFORM,‡ in the treatment of cardiac disease, while the important sedative and alterative actions of IODIDE OF POTASSIUM§ in syphilitic hypertrophy of the heart have been also pointed out, and its more important action in the treatment of

\* *Vide antea*, p. 188.

† *Vide antea*, pp. 306. and 309.

‡ *Vide antea*, p. 103.

§ *Vide antea*, p. 337.



aneurism will be fully treated of afterwards. I shall therefore conclude this lecture with a very few remarks on the importance of REST in the treatment of cardiac disease. Considering the difficulty most cardiac patients have in making any exertion, it seems almost superfluous to insist upon the importance of rest, yet it is a matter which cannot be too much insisted upon, and one which is only too apt to be neglected by many cardiac patients, who not infrequently vainly endeavour to benefit themselves by making ill-judged and injurious exertions, and who occasionally shorten their days by so doing. Whenever, then, from any cause there is defective, irregular, or in any respect uncomfortable action of the heart, and still more, whenever we have any uncompensated or imperfectly compensated valvular lesion, rest must be absolutely insisted upon as a necessary adjunct to the treatment, and this must be maintained for days or weeks, until the heart re-acquires a certain amount of its pristine vigour. In very many cases the mere act of getting out of bed and lying on a sofa for a few hours is too much for the patient, and delays his recovery considerably; while any greater exertion not infrequently thwarts all our endeavours to benefit the patient, and at all events renders our treatment less safe and certain. You must never forget, therefore, to insist upon rest as a most important part of the treatment, a rest which must be the more perfect and complete the more serious the cardiac disturbance is. Yet such patients are not to be confined to bed or to a Bath chair for all their future lifetime. You have constant opportunity of seeing that even the most strenuous exertions are not incompatible with the long-continued existence of very serious cardiac disease; and you have quite as frequently an opportunity of seeing that, after the cardiac disturbance for which the patient came under treatment is allayed, and the heart's nutrition improved by rest and appropriate treatment, he again acquires the capacity for exertion, at all events to a certain extent. We know that while the heart directs, and in some measure maintains the circulation, yet that various

subsidiary forces are of considerable importance in regard to this, and that muscular movement of itself, as well as by being a gentle stimulant to many of the secretions, is one of the most important agents in this matter. Hence gentle shampooing is a most important matter while the patient is confined to bed, and so soon as the heart has re-acquired sufficient power to enable it with some degree of comfort to discharge its own part of the duty, gentle exercise will be found to be very useful in improving the general health of the patient, and even in improving the condition of the heart. Hence you see that though rest is a most important therapeutic agent, yet there is a time when EXERCISE itself is not only not to be debarred, but may be made a most useful adjunct to our other treatment.

The treatment of cardiac dropsy may as a rule be safely enough entrusted to digitalis, to which in such cases we are in the habit of adding squill in moderate doses. But indeed digitalis is the most important remedy, and by appropriate dosage will be found to do all that we desire. Sometimes when there is considerable ascites forming part of the general dropsy present in such cases, the difficulty of exciting diuresis seems to be increased by the general compression of the kidneys, and in these circumstances it may be a question whether we should tap the patient or have recourse to purgation. I confess myself to a preference for tapping, which is so easily and painlessly done now-a-days; when the patient objects to this, however, we may purge him, and for the purpose, croton oil, elaterium, or a combination of the two in moderate doses at regular intervals of four hours until free catharsis is fully induced, and this repeated every second or third day, will be found to be a very useful treatment. Southey's drainage tubes are often of great service where there is much anasarca present, as their employment saves a good deal of time, and also keeps the bedding dry. The fluid in uncomplicated heart dropsy is simple serum, and never irritates the skin like that effused in kidney cases.

## LECTURE XV.

## ON MALPOSITION OF THE AORTA, DUE TO RICKETS, SIMULATING ANEURISM.

GENTLEMEN,—Some few years ago, a young man presented himself to me, stating that he had been about six months under treatment for supposed aneurism of the aorta, and that he was desirous of obtaining my opinion as to his present state and future prospects. Upon stripping this patient, inspection at once revealed that he had a distinctly abnormal pulsation between the second and third ribs on the right side, extending for about three-quarters of an inch to the right of the sternum; also that he had a slight scoliosis (lateral curvature) of the spinal column. Upon palpation, the chest was felt to expand slightly more freely on the right than on the left side; the abnormal pulsation was felt to be distinctly fluid in character, synchronous with the apex beat, and certainly not more forcible than it. Upon mensuration, the right side of the chest, at the level of the fourth rib, was found to measure a full inch in circumference more than the left; the right side measuring fourteen inches and a half, the left one thirteen inches and a half. Percussion revealed nothing abnormal, except a trifling dulness to the right of the sternum just over the abnormal pulsation and closely restricted to it. Auscultation likewise revealed nothing abnormal; the respiratory murmurs and cardiac sounds were all normal; while over the abnormal pulsation referred to nothing more was audible than



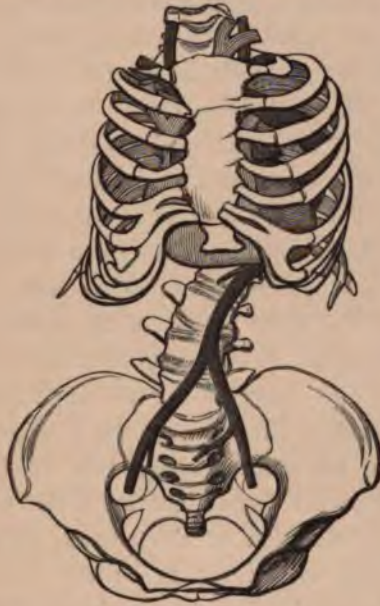
the sounds of the heart as they are normally heard at its base—left base, I was about to say—and, of course, I mean the base of the left heart; but, as you are aware, we auscultate the aorta at the right edge of the sternum. Moreover, there was an entire absence, not only of any signs of abnormal pressure, but even of any symptoms of such a thing. The young man was otherwise in perfect health, of very fair physical development, and quite capable of average exertion up to the time when he had been put under treatment. From a due consideration of all the facts elicited on examination, and thus cursorily narrated, I had no difficulty in telling the patient that he had no aneurism of any kind, that he was in perfect health, fit for any occupation, and only the subject of a very trifling malformation. I need not say how thankful and pleased he was. I have seen him repeatedly since that time, but have heard no more of his aneurism.\*

Now, this case is by no means a peculiar one; there are many such. Very possibly there may even have been some such cases whose lives have been embittered, or whose friends have been made unnecessarily anxious, by an unguarded prognosis; and to enable you to avoid this is the object of the present lecture. Hitherto these arterial malformations have been, at least of late years, almost entirely overlooked. Indeed, from a physician's point of view—that of diagnosis and prognosis—I may say that I know of no place where they have been referred to at all, except in a short paper by myself in the *Edinburgh Medical Journal* for February 1871, where I have related one case in which malformation of the aorta gave rise to the suspicion of aortic aneurism.† From an anatomico-pathological point of view, such cases have, however, long been well known; and though, as already stated, they have

\* This gentleman came to report himself a few months ago; his chest had considerably developed, and though the relations of the one side to the other remained the same (18 to 17), the artery was better covered, and pulsation was no longer to be felt. Sept. 1881.

† Cases Illustrative of some Difficulties in the Diagnosis of Aneurism close to the Heart.—*Ed. Med. Jour.*, Feb. 1871, p. 707.

been too much overlooked of late years, yet Morgagni,\* Watzel,† Vrolik,‡ Otto,§ Tiedemann,|| and others, have recorded many interesting cases; and in quite recent times the whole subject has been gone over from the same point of view by Dr



Barkow,¶ Professor of Anatomy at Breslau, who has figured a great many examples of this interesting peculiarity. One of the most remarkable of these is that contained in the fourth plate of his earlier work, in which the ascending aorta reached

\* *De Sedibus et Causis Morborum*. Ebroduni in Helvetia, 1779. 4.

† *De Efficacia Gibbositatis in Mutandis Vasorum Directionibus*. Trajecti ad Viadrum, 1778.

‡ *Dissertatio de Mutato Vasorum Sanguiferorum Decursu in Scoliosi et Cyphosi*. Amstelodami, 1823.

§ *Lehrbuch der Pathologischen Anatomie*. 1830.

|| *Supplementa ad Tabulas Arteriorum Corporis Humani*. Heidelbergæ, 1846. Hartmann, Cheselden, Ludwig, and Wenzel have also described these alterations of the vessels from a pathological point of view.

¶ *Die Verkrümmungen der Gefäße dargestellt* von H. C. L. Barkow, M.D. &c. Fol. Breslau, 1869. *Erläuterungen zur Lehre von den Erweiterungen und Verkrümmungen der Gefäße* von H. C. L. Barkow, M.D., &c. Fol. Breslau, 1871.



the surface of the chest between the third and fourth, and second and third ribs on the right side, and the descending aorta between the second and third ribs on the left side, the convexity of the scoliosis being to the left. The accompanying woodcut is reduced from this remarkable figure.

In the case just narrated, the scoliosis was so slight, there was probably slight malposition of the vessel itself, rather than any mere abnormal deflection of the artery due to the trifling curvature of the spine which existed. This however rather enhances the importance of the case from a diagnostic point of view. For while it is of consequence to remember that, in rickety chests, the aorta may be so curved as even in the absence of any marked dilatation, to make its pulsations visible either to the right or left of the sternum, and so to simulate an aneurism. It is of even more consequence to remember that in rare cases, similar abnormal pulsations may be visible from trifling alterations in the position of the vessel itself, apart from any marked changes in the bony skeleton. While we must never forget that an aortic aneurism may coexist with malformation of the thorax, with or without scoliosis, and that, whatever may be the condition of the skeleton, any abnormal pulsation must be carefully considered from every point of view before we even attempt to determine upon what it actually depends. For the art of diagnosis is based upon an estimate of probabilities, and its approximate accuracy depends, first, upon the accuracy with which we make our preliminary observations, and, second, upon the intelligence and capacity employed in our reasonings regarding these observations. For example, any cursory observer who had seen, but not duly considered, the case just related and who shortly afterwards was brought face to face with the woman Murray, now in bed 15, Ward XIII., would probably feel little hesitation—might even plume himself upon his sagacity—in placing them both in the same category; and yet he would thereby commit a most serious blunder. It is true that she has an abnormal pulsation between the second and third,



and the third and fourth ribs on the right side close to the sternum, associated with a diminution in size of the left side of the chest, which measures a full inch less in circumference than the right side. It is equally true that she has no scoliosis; but, in the young man's case, that was so slight as to be unimportant; and the diagnosis in either case turns entirely upon quite other points, to which I shall presently direct your attention. I shall first narrate to you the histories of two extremely interesting cases, in which aortic aneurism was very closely stimulated by vascular deformity depending upon rickets. Having already pointed out those negative signs which determined the diagnosis in the case first referred to, I shall then cursorily indicate those positive signs upon which we must rely for the definite diagnosis of aortic aneurism, a due consideration of which aided us in arriving at a very different conclusion in respect of the case of the woman Murray.\*

Case XXXIV.—G. S., a shoemaker, aged 35, admitted to Ward V., on 5th December 1870, complaining of breathlessness, cough, spit, and occasional hæmoptysis. When about 16 years of age, while acting as a cattle herd, he was occasionally troubled with slight rheumatic pains in the shoulders. But excepting a slight attack of ague (in Iowa, U.S.A), about two years ago, he has neither had rheumatism since nor any other ailment; he was 18 years of age, when he suffered from loss of appetite, and occasional squeamishness and faintness. He had then been engaged at shoemaking for about two years, he was well fed, and was able to keep at his work; just then however, his thorax became gradually deformed. On admission his spinal column had a double lateral curvature—the thoracic curve being to the right, and the compensatory dorso-lumbar curve to the left—while the lower dorsal and lumbar vertebræ were so twisted that the transverse processes lay somewhat diagonally; the ribs were broad and flat, the upper ones apparently wider apart than usual, the lower ones

\* This patient subsequently died, and her aneurism was exhibited at the Edinburgh Medico-Chirurgical Society, on May 3, 1876, *vide Ed. Med. Journal*, June 1876, p. 1141.

below the sixth crowded together. The anterior part of the thorax was also so altered that the right side was round and prominent, while the left was flattened and compressed. The lower part of the sternum was concave, as is so usually the case in cobblers. Both feet were twisted outwards, but he said they had been so from infancy.

About a year previous to admission, he began to have at times a severe pain across the front of his chest below the mammae. This pain usually came on while stooping over his work, but at times when he was otherwise engaged, occasionally even when in bed, and this it did sometimes for two weeks at a stretch, compelling him continually to shift his position, and even to get out of bed. He did not think that any change of posture gave relief. The pain came on suddenly, was not accompanied by any breathlessness, and it usually went away suddenly; he attributed it to the pressure of the boot on his breast necessary in his trade.

About six months previous to admission he first began to have shortness of breath, earliest felt on making any sudden movement, such as raising himself quickly from a stooping posture, but it speedily came to follow any exertion however slowly made, especially such as ascending a hill, &c. During the summer of 1870 he had considerable cough, and in August of that year when bathing, he brought up some blood mixed with the sputa; this trifling hæmoptysis lasting only one day at this time. His cough then became worse, and six weeks later his hæmoptysis recurred, lasting three or four days. About one month before admission he had a third attack of hæmoptysis, which lasted for three weeks.

*On inspection* of the thorax, the points already pointed out were seen, and considerable heaving of the precordial region was also noted; the spaces between the fourth and fifth and the fifth and sixth ribs being retracted at every diastole. Between the fifth and sixth ribs lay the lowest point of cardiac pulsation to be seen or felt, the ribs beneath this closed up, almost overlapped each other, and extended down

into the pelvis. This, however, was no apex beat, but a broad impulse diffused over a space of two inches and a half. The large arteries of the neck pulsated visibly with great force, and some thrill was felt at each pulsation. On laying the hand over the upper part of the thorax a considerable amount of thrill was felt, chiefly towards the right edge of the sternum and across its upper part. On putting the finger into the tracheal fossa the aorta was felt pulsating within half an inch of the upper edge of the sternum. Between the second and third ribs, on the right side, a pulsating tumour was to be felt, extending for about an inch to the right.

*On percussion* on the left side, one inch from the edge of the sternum, the percussion note was found to be clear down to the upper edge of the fourth rib; dulness extended from that to the upper edge of the sixth rib; and beneath this nothing was perceptible on percussion but the tympanitic note of the stomach. Dulness in the nipple line began about an inch to the right of the sternum, and extended across for a distance of four inches and three-quarters. Along the right edge of the sternum, from the upper edge of the second rib down to the liver dulness, for a distance of one inch to the right, the percussion note was dull.

*On auscultating* over the lowest part of the cardiac impulse, the first sound was heard somewhat muffled, and the second was replaced by a murmur. Between the second and third ribs at the right edge of the sternum, a loud, rough murmur replaced the first sound completely, and the second sound was also wholly replaced by a softer blowing murmur. Both of these sounds were louder and harsher over the pulsating tumour already mentioned, and became softer in character, though remaining equally distinct, on auscultating over the sternum. These bruits were propagated upwards into the vessels of the neck, and across the sternum, to the left. The pulmonary second sound was distinctly audible just over the left edge of the sternum close to the second interspace, within which it was only faintly to be heard close to the sternum.



The pulse was 86, full, and jerking, and was also delayed, the radial pulse coming just between two cardiac impulses, and as nearly as possible equidistant from both. On admission his cough was nearly gone, but he still had a slight amount of purely catarrhal expectoration. Other phenomena unimportant,—either natural, or without bearing on the case.

In this case the whole of the urgent symptoms and signs were those of incompetence of the aortic valves, a somewhat rare occurrence when the disease is mainly of the nature of a sacculated aneurism just above the valves. For when the disease has originally been a sacculated aneurism of the aorta the heart is rarely much implicated, even after the signs of aortic valvular incompetence have been superadded. Further, the signs present—especially the comparatively slight amount of dulness to the left of the sternum, and the greater amount of dulness to the right, and particularly the fact that the pulmonic second sound was scarcely audible at all to the left of the sternum, and only became so after we had placed the stethoscope upon that bone—all pointed to the great probability that the heart had been slightly dislocated to the right. But this, if correct, would of itself sufficiently explain the appearance of a pulsating tumour between the second and third ribs on the right side, and extending not more than one inch from its right margin, as this is just the situation in which the aorta might be expected to appear in such a case. The probability that the pulsating tumour was of this character was further increased by discovering from the augmented dulness across the upper part of the sternum, and the feeling of pulsation in the tracheal fossa, that the transverse portion of the aorta was dilated. Because all experience teaches us that in a case of aortic incompetence with a dilated transverse portion, dilatation of the ascending portion of the aorta is almost certain to coexist. But any dislocation of the heart to the right would suffice to push even a normal aorta from under cover of the sternum, and still more, therefore, one which is dilated. Besides, the slight increase in loudness of the abnormal

murmur in this case, was no greater than might be expected from the closer approximation to the ear of the pulsating tumour in the second intercostal space, as compared with an artery lying beneath the sternum, and wanted the distinct accentuation of such murmurs as heard over an aneurism. Moreover, all pressure symptoms were entirely absent. The lung on both sides was evidently displaced, and to its absence was unquestionably due the diastolic dimpling of the fourth and fifth interspaces on the left side; while the proximity of the aorta to the walls of the chest was undoubtedly the cause of the greater loudness and roughness of the bruit over the tumour, than over the sternum. The slight displacement of the heart was probably caused by the deformed condition of the chest due to rickets, the base of the heart being more displaced than the apex, as if the enlarged heart resting on the diaphragm in this deformed and stunted body leaned somewhat forwards and to the right into the bulging right half of the thorax; and this I have no doubt it did.

This patient died on 1st January 1871, from sudden œdema of the lungs; and though a proper examination of the body was refused by the friends, I was enabled satisfactorily to ascertain that, though the aorta was dilated and projected in the direction indicted, there was no saccular aneurism connected with it.

There can be no reasonable doubt that in this case the cardiac disease was primarily due to the obstruction opposed to the circulation by the sinuosity of the arterial trunks produced by the distortion of the skeleton, which, as Barkow has pointed out, has an unmistakable influence in promoting dilatation and hypertrophy of the heart.\* The increased energy of the heart in such cases has a special influence in producing dilatation of the aorta, especially of its ascending portion,† an influence which we can readily understand may be modified very considerably by the structural condition of the

\* *Die Verkrümmungen der Gefässe*, s. xv.

† *Op. cit.* s. xxxv.

aorta itself.\* When once dilatation of the ascending aorta is produced, the secondary development of incompetence of the aortic valves is merely a question of time.

The next case was repeatedly under observation, and was always an object of great interest.

Case XXXV.—M. P., a milliner, unmarried, aged 46, admitted to Ward XIII., on 26th January 1871, complaining of cough, feeling of oppression over the trachea, and of general debility. She had been a milliner for thirty-three years, working during the busy season from 6 A.M. of one morning to 1 A.M. of the next. As the result of this overwork she was never strong, and never free from headache; still, she could take her food well, and was able to continue her work. About sixteen years before admission, she first perceived that her right shoulder was somewhat distorted. Since that time this, as well as the distortion of her body, which she also then first noticed, had gradually increased. But the change had been gradual, and she suffered from no illness more severe than a mere casual and temporary catarrh, till seven years before admission, when she suddenly found herself affected with a cough, which has never since entirely left her. She was not ill nor feverish at that time, and was unconscious of having caught cold. Two years subsequently, however, this persistent cough was aggravated by an attack of bronchitis, which lasted for five weeks, and broke down her health very much. Since that time her bodily distortion had steadily increased; and about a year after this her bodily distortion and debility became so great, that she was compelled to give up her occupation. Since then she has been constantly subject to a dull aching pain, referred to the right shoulder-blade, the intensity of which has gradually increased. She has also been subject to pain in the region of the stomach, more or less constant, but worse when the stomach is empty, and somewhat relieved by taking food.

For some years she has been aware of a pulsation in the

\* *Op. cit.* s. xv.



front of her neck, but her attention was not particularly directed to it till the morning of the 23d of January 1871, when, after a severe fit of coughing, she accidentally observed that where this simple pulsation formerly existed there was now a throbbing swelling equal in size to a hen's egg. A feeling of tightness and choking over the chest and lower part of the throat, which subsisted after the cessation of the paroxysm of coughing, induced her to look at her throat, and so to discover this pulsating tumour. On admission, the patient was seen to be much emaciated and etiolated; there was considerable scoliosis of the spinal column in the dorsal region, with right side convexity, some protrusion backwards, and compensatory lumbar curve to the left; the thorax on the right side was thrown outwards and backwards, and flattened laterally; on the left side the ribs were indented and compressed together.

On palpation, the only pulsation to be felt on the left side was between the third and fourth ribs, about half an inch to the left of the sternum. To the right of the sternum pulsation was felt in the first, second, and third interspaces. This pulsation was most forcible and distinct in the second interspace, where it extended a couple of inches to the right of the sternum, and in this situation a considerable thrill was to be felt. In the lower part of the neck, just over the suprasternal notch, a pulsating tumour, evidently a dilated artery, was felt crossing the trachea and dipping beneath the sterno-cleido-mastoid muscle, being apparently continuous with the right subclavian artery, which, as well as the brachial, was large and dilated. The left subclavian was also dilated. The aorta was not felt on passing the finger deep into the tracheal fossa. All the arteries at the root of the neck pulsated strongly.

On percussion, the cardiac dulness on the left side was found at one inch from the sternum on the left side, to commence at the upper border of the second rib whence it extended down to the liver dulness. From the upper border of the second rib,

dulness across the sternum gradually rose to the lower border of the first rib, and at the right edge of the sternum this dulness passed outwards between the first and second ribs to a distance of two inches from that bone, and it also passed downwards to the liver dulness in the same parasternal line. At the level of the fourth rib the transverse dulness was four inches and a half. On auscultating over the cardiac pulsation to be felt between the third and fourth ribs, half an inch to the left of the sternum, both sounds were to be heard, neither very pure. Over the fifth rib, one inch from the left edge of the sternum, these sounds were to be heard with greater distinctness and purity, but no pulsation was to be felt. In the second interspace to the left, distinct but impure, first and second sounds were both to be heard. In the second interspace to the right of the sternum the first sound was impure and the second was obscured and almost entirely replaced by a diastolic murmur. Over the pulsating tumour already referred to as chiefly lying between the first and second ribs to the right of the sternum, where the pulsation was most fluid and most forcible, we had a loud systolic murmur, followed by a less distinct diastolic murmur. These murmurs obviously had their position of maximum intensity in this situation, and from it both radiated outwards in all directions; the systolic murmur extended upwards with most distinctness, the diastolic murmur was propagated with more distinctness downwards, but *pari passu* less distinctly, being more faint *ab origine*. The pulmonary percussion was equal on both sides, and the respiration also equal, though neither could be held to represent an average normal, as the one was slightly higher in the pitch, and the other slightly rougher in character. No other signs or symptoms of any importance could be elicited, except that over the pulsating tumour already referred to considerable systolic thrill could be perceived.

Obviously the diagnosis in this case was not far to seek; the absence of the slightest sign of any abnormal pressure, in spite of the existence of an abnormal pulsating dulness in

the first interspace to the right of the sternum, was quite conclusive as to the non-existence of any sacculated aneurism, or even of any considerable arterial bulging of a cirroid character in that region, and equally conclusive as to the pulsation being simply arterial in character, this being further confirmed by the strict limitation of the dulness to the pulsation. But a pulsating arterial tumour in the region referred to could only be aortic in its origin; and, if only a simple dilatation, must be of unprecedented dimensions to present so large a superficial area of dulness. The position of the cardiac pulsation, however, as well as the situation and form of the area of cardiac dulness, assured us that the heart was tilted upwards as well as thrown more to the right of the sternum than usual; hence we had the ascending portion of the aorta passing more directly outwards to the right; and, as the transverse portion of the arch did not rise much higher nor take a wider sweep than ordinary, it must necessarily have made, with the ascending part, a more than usually acute angle. Hence, of course, compression of the lumen of the vessel at this acute angle, and a complete explanation of the systolic thrill and the loud systolic murmur, necessarily due to the formation of fluid veins where the blood passed through the constricted part of the artery into another portion, which was possibly more dilated than usual, although that was by no means essential. As for the diastolic murmur, in the absence of any saccular aneurism, which we believed not to exist, its only possible cause must have been regurgitation through the aortic valves, the result of the increased pressure due to accumulation of blood in a dilated ascending aorta. The effect of this in every case must be ultimate separation of the segments of the valve from hydraulic pressure, an inevitable physical result which might be complicated and hastened by the certainly less inevitable physiological results of pressure—local endocarditis, thickening and shrivelling of the valve.

This patient died suddenly from syncope, in Ward XIII., on 7th November 1872. At the autopsy the lungs were found



congested and œdematous. The heart was tilted up, and the great vessels were displaced. The ascending aorta passed outwards to the right more than usual, and the transverse portion passed off from it at a somewhat acute angle. The innominate was two inches long and twice its usual diameter. It came off from the aorta further to the left than usual, coming to the surface at the left sterno-clavicular articulation, and passing across the trachea in the lower part of the neck to the edge of the right sterno-cleido-mastoid muscle, beneath which it dipped and divided. The aorta was slightly atheromatous and dilated at its commencement, but perfectly free in every part from any saccular enlargement; its valves were much thickened and slightly incompetent. The heart itself was hypertrophied and dilated, particularly on its right side, but to no great extent. The mitral valve was competent, but had a few vegetations on its upper surface. The tricuspid valve was healthy; its opening admitted five fingers. The pulmonary valves were healthy and competent. The abdomen was filled with fluid, the liver was slightly enlarged, and the kidneys in a state of chronic congestion. No other organ was examined.

Here, then, you see we had another well-marked example of rickety distortion of the skeleton giving rise to abnormal conditions of the blood-vessels of such a character as very closely to simulate aneurism. Of this peculiar simulation, I think this latter case may be considered rather an extreme example; and between it and the case with which I commenced this lecture, there are infinite gradations, examples of which are of occasional, though not of very common, occurrence. It is well to be aware of, and prepared for, the occurrence of such cases, and thus avoid falling into mistakes, which, in any case similar to that I have just referred to, might be fraught with very serious results to one or other, possibly to both, of the parties concerned.

You will, therefore, remember that even a fluid pulsation in any of the intercostal spaces is not necessarily an aneurism. That the absence of any history of empyema, or even the dis-

tinct connection of the pulsation with the aorta by continuity of dulness, is no proof of its being aneurismal in character, either in the sense of a sacculated aneurism, or of its being a simple dilatation. Because, even in the normal condition of the skeleton, the aorta may exceptionally be so deflected as to cause its pulsations to become perceptible in one or other of the intercostal spaces; while abnormal intercostal pulsation of simple arterial origin is a matter of no infrequent occurrence whenever the thoracic skeleton is deformed by rickets; we must also never forget that, even in chests deformed by rickets, sacculated aortic aneurisms may occur.

Where there is no twisting or bending of the artery, and no aortic regurgitation, the first case narrated would seem to show that—as we would naturally expect—there is no murmur to be heard over the abnormal pulsation, and that the only sounds audible are those ordinarily heard at the base of the heart, the second, in particular, not being in any degree accentuated. But whenever we have a murmur of regurgitation developed at the base of the heart, that is always more or less audible over the course of the ascending and transverse portions of the aorta. Apart, also, from any constriction at the mouth of the aorta, we are sure to have a systolic murmur developed over any part of that artery where any sharp twist or bend occurs. Moreover, as any abnormal intercostal pulsation of the aorta must be nearer the surface than any normally situated part of the artery, all sounds in its course are to be heard louder over the pulsation than elsewhere, yet without accentuation. But whenever we have marked accentuation of these sounds, and especially if the normal cardiac sounds, particularly the second, be accentuated, we must suspect the occurrence of a sacculated aneurism, and that even though rickety malformation of the chest be present. To make our diagnosis certain, however, we must be able not only to connect the pulsation directly with the aorta, but to show also that the dulness subtended by the pulsating body occupies a space greater than would be the

case were the pulsation due to a simple cylindrical vessel such as the aorta. Extension of dulness, therefore, beyond the pulsating tumour, associated with the signs and symptoms of pressure upon one or other, or upon several of the neighbouring organs, are among the most certain indications of the existence of a sacculated, or even of a cirroid, aneurism. And from these signs and symptoms of pressure, associated with certain other phenomena which indicate the dependence of that pressure upon an elastic and distensile body of varying dimensions, we are often able to prognosticate the existence of an aneurism, even when no pulsating tumour has been detected; while a due attention to the phenomena present will enable us to determine with almost perfect accuracy the exact nature of any pulsating tumour which may be perceptible. But I must reserve the full consideration of this matter for next lecture.



## LECTURE XVI.

## ON THE DIAGNOSIS OF AORTIC ANEURISM.

GENTLEMEN,—The diagnosis of thoracic aneurism is sufficiently obscure at times, while at others it seems so patent that it is difficult to conceive the possibility of a mistake, yet even in cases apparently the most simple, mistakes are not only possible, but are occasionally made by men of considerable experience, not only as to the nature of the affection, but quite as frequently as to its seat; aortic aneurisms being not infrequently sent to hospital to be ligatured, a procedure of course inadmissible.

I need hardly say what an aneurism is, that it is a local dilatation of an artery, of all its coats in a fusiform or globular shape, or merely a bulging of these coats to one side or the other, these two forms being often united in the thoracic aorta, in which uniform dilatation is frequently associated with local bulgings. The form caused by uniform dilatation of all the coats is termed a *true aneurism*. The bulgings are, however, frequently entirely local and circumscribed, unaccompanied by any general arterial dilatation, but, on the other hand, accompanied and apparently produced by rupture of one or other of the arterial coats; this is what is termed a *false aneurism*. It is frequently also called a *sacculated aneurism*; but it is well to remember that this term is also occasionally applied to the bulgings already described as occasionally associated with uniform dilatation, especially when these are unilateral. During life it is scarcely possible to state, even

with a minimum of probability, whether a sacculated aneurism is true or false; but it is quite possible to say, with great probability, whether it is associated with dilatation of the vessel, and therefore probably a mere bulging, or whether it stands alone, and is therefore all the more likely to be not only a sacculated but also a false aneurism. These false aneurisms have been pathologically subdivided into several varieties, according to the number and nature of the coats ruptured: clinically this is a matter of no importance, and impossible to recognise. So-called *dissecting aneurisms* are those in which the two inner coats are ruptured, and the blood effused between them and the outer coat. The accurate diagnosis of this event is not easily made; fortunately it is of but slight importance in relation to treatment, though of some consequence to prognosis. *Varicose aneurisms* chiefly affect the arch of the aorta, and consist of abnormal communications between it and the superior vena cava, the pulmonary artery, or the right auricle. These give rise to phenomena of great pathological interest, are usually rapidly fatal, and are not amenable to treatment. *Cirroid aneurism* of the aorta, or what is called so, is by no means rare, at least if we take Rokitsansky's definition of this form. He says that any cylindrical or fusiform dilatation of the artery, accompanied by apparent increase in length of the vessel, and by bulgings first on one side and then on the other, so that the vessel winds, as it were, from side to side, and lies in apparent coils, is entitled to this appellation.\* A great number of what are termed true aneurisms of the thoracic aorta present this appearance, and are therefore entitled to be called cirroid, though they have nothing in common with the external so-called aneurisms usually recognised by this term.

Again, when we speak of aneurisms of the aorta we usually refer to those occurring above the semilunar valves, but it would be wrong to regard them as the only aneurisms of the aorta. For, besides aneurisms of the valves themselves—which

\* *Handbuch der Pathologischen Anatomie*, 1844. II. Bd., s. 551, 577.

ought perhaps to be looked upon as actually belonging to the aorta, but which, when they reveal themselves clinically at all, do so only as valvular lesions—we have also, though only rarely, aneurisms situate immediately within the valves and above the cardiac ventricle (intra-avalvular aneurisms), and aneurisms between the valves (intervalvular aneurisms), the latter being much the most rare; but both forms are exceedingly uncommon.\* The symptoms during life, so far as observed, were only those of valvular lesion; clinically, therefore, they possess no peculiar interest, though they are of considerable pathological importance.

True aneurisms—simple dilatations of whatever form—whether accompanied or not by bulgings, and false or saccular aneurisms, arising above the valves, are those of the greatest clinical importance, and they are much more frequent in the thoracic aorta than in any other part of its course. This we learn conclusively from Dr Sibson, who at a great deal of personal trouble has collated the records of 584 cases of aneurism, and also examined 296 specimens in museums, to which no histories are attached—880 cases in all.† From his researches the following table has been compiled:—

Situation of Aneurism.	Total Number	Of these there were Sacculated.	Mere Dilatation of Vessel.	Character of Aneurism not defined.
Sinuses of Valsalva . . . . .	87	95·95	3·56	
Ascending Aorta . . . . .	141	54·75	38	7·25
Do. Dissecting Aneurism . . . . .	52			
Transverse Aorta . . . . .	120	44·25	20	12·5
Ascending and Transverse Aorta conjointly . . . . .	112	40·7	85·7	
Descending Portion of Arch . . . . .	72	71·9	14·55	17·7
Transverse and Descending Portion of Arch . . . . .	20			
Whole Arch . . . . .	28			
Descending Thoracic Aorta below Arch . . . . .	71	42·4	42·55	20·4
Abdominal Aorta at Coeliac Axis . . . . .	131	70·35	11·85	18·4
Do. below Mesenteric Artery . . . . .	26			
Branches of Abdominal Aorta . . . . .	20			

\* *Vide* three cases, two of the former and one of the latter form, related by Rokitsansky, in the *Medizinische Jahrbücher*, s. 174. Wien, 1867.

† *Medical Anatomy*. London, 1869: Churchill & Sons. Columns 57 and 58.



Thus of 880 cases, no fewer than 703 belong to the thoracic aorta, and if we take any one portion of its course we find that by far the largest number of aneurisms belong to the ascending aorta, which includes no fewer than 193, 52 of them being dissecting aneurisms. The transverse portion of the arch is much less frequently involved, its aneurisms numbering only 120 out of the 880, while aneurism of the descending portion of the thoracic aorta is still more rare, only 72 out of the 880 belonging to that portion of the artery. Further, if we take the number of aneurisms belonging to the ascending portion of the aorta and to its transverse portion conjointly, and compare them with those arising from the transverse and descending portion of the arch conjointly, we find the relative numbers to be 112 and 20. All these facts tend to show the great importance of the ascending portion of the aorta in relation to aneurism. The cause of this is purely mechanical, and arises from the strain of the cardiac impulse being necessarily most felt in this situation, on account of the relation of this portion of the artery to the left ventricle.

The middle coat is that upon which the strength, firmness, and elasticity of the arterial tube depends, and loss of elasticity in it must precede and is the primary cause of arterial dilatation or aneurism, the secondary and effective one being of course the intra-arterial blood pressure. Localised bulgings and saccular aneurisms are due to localised atrophies or ulcerations of the middle coat. These affections are degenerative in character, and are usually described as following a primary affection of the *intima* (chronic endarteritis); sometimes, however, the *media* itself is primarily affected, and more rarely aneurisms have been found to follow a circumscribed inflammation implicating the external and middle coats (periarteritis nodosa), or the impaction of a sharp pointed embolus; while falls, blows, and violent exertions are often the direct cause of the formations of an aneurism, by rupturing the coats of a vessel already diseased.

There is no disease the diagnosis of which is more beset with difficulties than thoracic aneurism, but there is also probably none in which a due consideration of all the physical signs and symptoms, as well as of their modification by position, exertion, &c., and of the mode in which they have been primarily developed and have subsequently progressed, is more capable of conducting us to a satisfactory, if not always a perfectly positive, diagnosis. There is only one phenomenon positively characteristic of thoracic aneurism, and that is *the existence in some part of the thorax of a pulsating tumour other than the heart, which beats isochronously\* with it and at least as forcibly, and which at each pulsation expands in every direction*. These signs distinctly recognised are sufficiently distinctive, and yet they are occasionally so efficiently simulated as to necessitate great care in their determination as well as recourse to subsidiary assistance. A solid tumour lying on the aorta may give rise to a bruit, and may apparently pulsate, but it only rises and falls with the beating of the artery, and does not expand in every direction. A vascular tumour, be it mediastinal or pulmonic, similarly situate, may possess not only a bruit but also a certain degree of expansive pulsation; that pulsation, however, is not *isochronous* with the heart's action, but always somewhat delayed. An empyema may present both bulging and expansile pulsation isochronous with the heart, and, when subcutaneous, or even when more truly intra-thoracic, may efficiently enough simulate an aneurism; in these cases, however, the pulsation, though expansile from being communicated through fluid, is less forcible than that of the heart; there is also absence of thrill or murmur, with the existence of the other signs, symptoms, and history of an empyema.†

\* In one case I found the pulsation, though thoracic and truly aneurismal yet not isochronous, but this exceptional case does not invalidate the above statement.

† Vide an interesting paper by Dr M'Dowall in the *Dublin Quarterly Journal* for March 1844, on "The Diagnosis of Empyema," especially at page 16, where the diagnosis between pulsating empyema and aneurism is considered.



I have already narrated a series of cases in which uncovering of the pulmonary artery gave rise to visible pulsation over which a systolic bruit was audible, simulating thus very closely an aneurism, and I then pointed out the diagnosis between the two.\* We also not infrequently have aneurism simulated by uncovering of the aorta, which in some such cases is perfectly normal, though more usually somewhat dilated as well as bent or twisted,—the result of deformity of the spine—scoliosis—and distortion of the thorax. I have already given you the details of several such cases,† so that we need not enter upon their history further at present.

The importance of these cases in relation to the diagnosis of aneurism in this situation is sufficiently evident. The points of resemblance are also quite apparent. The points in which they differed were—first, in the entire absence of all the subsidiary phenomena dependent upon pressure on the neighbouring organs, phenomena which are often obscure, and to the consideration of which we shall presently recur; and, secondly, in the fact of the isochronous pulsations being less forcible than those of the heart. That aneurismal pulsations are usually more forcible than those of the heart is a diagnostic point to which attention has not hitherto been paid. It first occurred to myself as a matter of some consequence in the diagnosis of aneurism in relation to the cases just narrated, as well as others of a similar but less delusive character. On talking the matter over with Dr Henderson, late Professor of Pathology in the University of Edinburgh, a distinguished authority on the subject, and himself a sufferer from the disease, I found that this had also struck him as an important point in relation to the diagnosis of aneurism close to the heart, and he believed he had pointed it out in his writings. I have, however, been unable to discover any reference to it in them. On the other hand, various writers on the subject,

\* Lecture VIII., p. 208.

† Lecture XV., p. 355.



among whom I may mention Dr Greene,\* have referred to it as a remarkable fact, without giving any reason for it, or any estimate of its diagnostic importance. Other writers, however, in narrating cases of indisputable aneurism, have mentioned that the pulsation in the sac was less distinct than that of the heart. The explanation of this discrepancy is, I believe, not far to seek: in order that the law I have mentioned (if it be a law as I believe) may hold good, the pulsations must be fluid; if the sac be lined with fibrine, other elements are introduced which neutralise those in force when fluids only are concerned. In substernal aneurisms—those close to the heart—fibrinous coagula are rarely present, and thus the physical conditions upon which this law depends usually remain in force just where it is of most importance that they should.†

Whenever, therefore, we have a tumour in the chest which pulsates isochronously, and at least as forcibly as the heart, we have certainly to do with an aneurism, which, if it does not now present, has probably at some previous period presented, other subjective symptoms, of which pain is that most apt to disappear during its progress; the signs of pressure, on the other hand, are more constant and more likely to increase, if we except the signs of pressure on certain veins, such as the brachial, which may entirely disappear as the aneurism progresses.

PAIN is a symptom which is most apt to be loudly complained of, yet inasmuch as it may arise from neuralgia (rheumatic) of the external coverings or from angina, it is not of itself much to be depended upon as a sign of aneurism; and therefore, while it ought always to direct our attention to the parts, as it must necessarily do, it can never of itself be accepted as an efficient diagnostic sign of aneurism, though we

\* *Vide* his paper on "The Symptoms and Diagnosis of Thoracic Aneurism," in *The Dublin Journal of Medical Sciences*, 1837, pp. 233 and 236.

† Allen Thomson says the pressure within an aneurism is equal to the extent of its internal surface multiplied into the force of the blood in the part of the artery from which it springs.—*Cyclop. Anat. and Physiol.* vol. i. p. 663.

may often find it a most valuable warning. The pain of commencing aneurism will be found to be more neuralgic and lancinating in character, as well as more continuous, than that of angina, and not usually so dreadfully oppressive and depressing; ranking also with neuralgic pain in this, that it often disappears for months without any very obvious reason. When the aneurism affects the large vessels at the root of the neck, or the aorta in this situation, the pain shoots across the chest as well as up the one side of the neck, or down one arm, or extends in both directions, sometimes shooting down both arms. When it affects the descending portion of the thoracic aorta it is usually referred to one spot on the back, where it is constant; though even in such cases sharp stings may shoot up the neck or down the arms. But in these cases we have usually both circumscribed dulness and localised pulsation, with or without bruit, and usually other symptoms which serve to confirm the diagnosis. And it is chiefly in incipient substernal aneurisms close to the heart that pain is so isolated and important a symptom as almost to entitle it to be called "pathognomonic," as Dr Greene has put it; yet a careful watching even of such cases will ere long elicit other symptoms which are more trustworthy and more truly pathognomonic of aneurism than pain alone, which is really only an important warning, but a warning with a query. Pain in the situations referred to is chiefly produced by pressure on the intercostal nerves, or on those of the brachial plexus, and a constricting pain around the lower part of the chest is occasionally produced by pressure on the phrenic nerve; pressure on the latter nerve also giving rise occasionally to attacks of dyspnœa, singultus, or even to complete paralysis of the diaphragm. Pressure on the pneumogastric, besides giving rise to disease of the lungs, in its earlier stages is frequently accompanied by vomiting, or severe dyspepsia, accompanied by flatulence, often relieved by gentle rubbing of the tumour.

DYSPNŒA is a very frequent symptom of aneurism; it arises from compression of the trachea, one of the bronchi or



a portion of lung, or of one of the recurrent laryngeal nerves. When one of the recurrent laryngeal nerves is affected, the dyspnoea may arise from spasm, or paralysis of the vocal cords. In the former case both the inspiration and the expiration are affected; in the latter the inspiration alone is impaired. The voice in such cases is altered (*vox anserina*, shrill whistling voice); more rarely there is complete aphonia; and inspection by means of the laryngoscope reveals on which side the affected vocal cord lies. The left recurrent winding round the arch of the aorta in the neighbourhood of the origin of the left subclavian is the one usually implicated, the right recurrent passing round the right brachial artery being rarely affected by any aneurism. If the glottis is found to open freely, and the arytenoid cartilages to retain their normal movements, we know that any dyspnoea present is not laryngeal in its origin, and probably depends upon compression of the trachea or of one of the bronchi. When the respiration is found to be diminished equally on both sides of the chest, we are justified in regarding the trachea as the part compressed, and in such cases the transverse portion of the arch is usually at fault, though the trachea may be compressed, exceptionally, by aneurisms both of the ascending and descending aorta. On the other hand, compression of one of the bronchi gives rise to diminution of the air in that part of the lung to which it is distributed, over the whole of the one lung if it be one of the main divisions, over the corresponding part of the lung if it be one of the smaller bronchi. This diminution of the air in the lungs gives rise on percussion to a note of a higher pitch and more tympanitic character over the part affected,\* and it may give rise either to diminution of the respiratory murmur over the corresponding part of the lung, or more rarely to bronchial respiration,† which is a very remarkable phenomenon when associated as it is in such cases with a clear tym-

\* Skoda, *Abhandlung über Perkussion und Auskultation*, Wien, 1844, s. 18.

† *Vide* a case of Dr Halliday Douglas, *Edinburgh Medical Journal*, December 1869, p. 550. Dr Greene, *loc. cit.* p. 233. And for an explanation of this phenomenon, *vide* Skoda, *op. cit.* s. 108.



panitic percussion note. Dyspnœa arising from aneurismal compression of the trachea or bronchi is naturally increased by exertion, and frequently much relieved by posture, as by leaning forward when the pressure is on the trachea, or leaning to one side when one or other of the bronchi is affected. In such circumstances the air is heard to enter more freely than under the ordinary conditions, and thus postural aids to diagnosis are often of service in ascertaining the exact position of the aneurism. But dyspnœa may also arise from pressure on the lung itself; it is then frequently associated with symptoms simulating phthisis, and for these alone the patient occasionally seeks relief, wholly unconscious of his actual malady. Still more rarely the dyspnœa is brought about by compression of the pneumogastric nerves, which may either give rise ultimately to serious and fatal lung disease, or may of itself prove fatal by producing suffocation. Of all the organs within the chest, the lungs are those most frequently compressed by aortic aneurism, and this is the reason why a circumscribed dull patch is one of the most frequent signs of this affection.

COUGH is a very frequent symptom of thoracic aneurism, and as it arises most frequently from irritation of the pneumogastric and laryngeal nerves, it usually presents certain peculiarities, which, if not exactly pathognomonic, are at least sufficiently striking to excite attention. In such cases the cough is often loud and barking, accompanied by metallic clang. The cough is very distressing both to patient and bystanders, and appears far in excess of the necessities of the case, being accompanied, at first at all events, by only a small amount of glairy and frothy mucus. From persistence of the irritation the expectoration may, however, become more copious, and muco-purulent. When the cough arises from pressure on the lungs themselves, the expectoration becomes very early copious and muco-purulent, simulating that of phthisis. At times it may become rusty, or even red, and this betokens a very hazardous condition of the aneurism.

At other times, in that variety which is termed a weeping aneurism, the expectoration, without being copious or even accompanied by much distressing cough, always contains some small amount of fluid blood.

DYSPHAGIA is not an uncommon symptom of thoracic aneurism, and varies in extent from slight difficulty in swallowing, arising apparently from some interference with the œsophageal innervation, to that more complete form of dysphagia depending upon compression of the œsophagus in which there is either great difficulty in swallowing solids, or complete inability to do so. The diagnosis between this form of dysphagia and that arising from organic structure is usually readily made, inasmuch as organic dysphagia is unvarying and persistent, while aneurismal dysphagia varies from time to time—is complete at one period of the day, and wholly absent at another. It is also increased by any excitement of the circulation, and markedly relieved by position; thus a patient afflicted with aneurismal dysphagia may be able to swallow with tolerable freedom when he leans forward so as to free the œsophagus from pressure. I need scarcely caution against the great danger of using a probang in such cases; yet it seems not unlikely that in obscure cases a properly constructed and carefully introduced probang might assist the diagnosis by revealing pulsation, while even a bruit might be audible through it.

PRESSURE ON THE BLOOD-VESSELS produces various well-known symptoms, amongst which has been reckoned alteration of the radial pulses, so that one differs from the other in size or volume. We must, however, ascertain that this difference extends to the brachial, to avoid being misled by simple irregular distribution of the radial. The best mode of ascertaining whether this variation between the two radial pulses is congenital, or dependent upon aneurismal pressure, is to be found in the differences visible in the sphygmographic tracings of the pulse movements of each artery. This alteration of the radial pulse is, however, rarely if ever due to pressure,



being usually (invariably ?) dependent upon stenosis of the brachial artery, from a calcareous ring or projecting plate. Various head symptoms are, however, produced by interference with the cerebral circulation, headache, quasi-apoplectic seizures, &c. Pressure on the brachial veins produces swelling of the corresponding arm, unaccompanied by any signs of inflammation. Its dependence on aneurism is made probable when it comes on suddenly during any violent exertion, and especially if accompanied by any bruit localised in the innominate, brachial, or carotid arteries. In such cases the swelling occasionally disappears after a time, and this disappearance may be accompanied by the appearance of a pulsating swelling in the upper part of the chest. Considerable compression or even obliteration of the superior vena cava has been occasionally observed, and such cases are attended by a great development of the superficial veins. A moderate degree of this symptom is of no infrequent occurrence, even when the compression is not so great; a thick œdematous collar covered with enlarged veins surrounding the root of the neck in such cases. But in all these cases there are always plenty of other symptoms to lead us at once to the true diagnosis. Compression of the vena cava decedens or right auricle may give rise to congestion and dropsy, but these are usually late symptoms.

PRESSURE ON THE HEART by aneurism is a frequent cause of its displacement. If the tumour affect the ascending portion of the aorta, the heart is displaced downwards and to the left; if it affect the arch, the displacement may be solely downwards; and when the descending portion is affected, the heart may either be displaced to the right, or if the tumour be seated just behind the heart, the latter is compressed against the anterior wall of the thorax, and gives a larger and more forcible impulse. Consecutive alterations of the heart in aneurism are rarely of any importance; even when a double bruit at the right base reveals an alteration of the aortic valves, which is most likely to be attended by great con-



secutive changes, these as a rule are so slight as to be unimportant, and have little ultimate bearing on the history of the case. The pressure of an intra-thoracic aneurism may even be so great as to depress the liver several inches, and communicate to it a well-marked pulsation, and of this I have seen one well-marked example.

When the pressure of an aneurism affects THE NERVES ISSUING FROM THE SPINAL CORD, in what has been termed the "cilio-spinal region," that is, according to Budge and Waller, from the sixth cervical to the sixth dorsal nerve, or, according to Brown-Séquard, as low as the ninth or tenth dorsal nerve, from the anterior roots of which filaments pass through the cervical sympathetic to the iris, then certain peculiar phenomena are observed, according to the amount of pressure exerted. When the pressure is considerable, permanent contraction of the pupil on the affected side is the result; but when it is just sufficient to excite and not to paralyse the nerves, dilatation ensues. When contraction is extreme, the effect is very striking, but there are a multitude of cases in which the difference between the two pupils is comparatively slight, and in which it is scarcely possible to say which pupil is contracted, and which is dilated. In these cases we must determine by careful measurement which is the contracted pupil, and ascertain by the reaction to atropine, always incomplete in *spinal* myosis, and also by the insensibility of the pupil to the action of light, though it contracts still further on accommodation for near objects, that we have actually to do with a morbid condition, and not merely with an unusual but perfectly healthy state of matters, about one person in fourteen having one pupil naturally smaller than the other. Of course it is only on the left side that alterations of the pupil are likely to be produced by aneurism, and even when found there, these alterations are only indicative of a spinal lesion the cause of which has still to be discovered. Spinal myosis is not pathognomonic of aneurism, though it often helps us to detect one by the careful investigation of the region im-

plicated, as well as of the other phenomena connected with this peculiar phenomenon.\*

CIRCUMSCRIBED DULNESS is always present whenever the aneurism reaches the walls of the thorax. From the more or less globular shape of an aneurism, the tumour is invariably larger than the area of complete dulness. Most frequently the dull patch is found on the right side of the sternum, on a level with the second or third ribs, more rarely on the left side of the sternum, or over that bone itself, or at the back. In the latter case the aneurism will be found to arise from the posterior portion of the descending aorta, and it only rarely passes to the right of the vertebral column. Over this dull patch we always have more or less pulsation, which is more or less fluid and forcible in character, according to the thinness of the walls of the sac and the amount of clot contained in it. As the disease progresses, the circumscribed dulness increases, the walls of the chest become involved in the tumour, which finally breaks through and appears on the surface as a pulsating swelling, in which condition its character is scarcely to be mistaken. Over the dull pulsating tumour various SOUNDS are audible, and these vary with each case. Perhaps what we ordinarily understand by a murmur is comparatively the rarest phenomenon audible over such a tumour in the chest, a systolic jog or shock being all that is appreciable in many such cases, though occasionally this jog is double. In such instances, however, it has seemed to me, from the firmness of the impulse, that it was probable the tumour was well coated with fibrine internally, but this is certainly not always the case. Most commonly we have the normal sounds of the heart propagated over the tumour, and in this case it has been long known, having been first pointed out in 1836 by Dr Henderson, that the second sound, or that produced by the closure of the aortic valves, presents what he has called

\* Vide Dr Gairdner, in the *Edinburgh Medical Journal*, January and August 1855; Dr Ogle, in the *Medico-Chirurgical Transactions*, vol. xli.; and for the physiology of the subject two interesting papers by Dr Argyll Robertson, in the *Edinburgh Medical Journal*, February and December 1869.

"a very striking resemblance to the shutting of a pump-valve in the immediate vicinity of the ear,"\* and which has been variously designated by succeeding observers as a ringing, booming, or accentuated second sound. This accentuated or booming sound, when heard over a circumscribed dull patch, and limited to that position, is very distinctive of aneurism. If, however, it be produced so near as to be readily propagated to the aortic area, it may be confounded with a similar accentuation audible in that area when we have a dilatation of the ascending aorta.† Heard out of the aortic area, therefore, and over a circumscribed dull patch, it is almost distinctive of aneurism; heard within that area, it may be due to dilatation of the aorta, but if, under these circumstances, it is heard propagated to the right or the left of the usual course of the aorta, the probabilities are in favour of dilatation with bulging (cirroid aneurism), rather than of a true saccular aneurism. When there is a double murmur at the base of the heart, the same murmurs, only intensified, are heard over the tumour. At times a systolic murmur of varying character, preceding the accentuated second sound, is heard over the aneurism, and still more rarely we have also a loud diastolic murmur. In the only case in which the latter occurred in my own experience, the opening of the aneurism was large and smooth, and it was difficult to account for the occurrence of a diastolic bruit at all. A localised and circumscribed murmur anywhere in the course of the aorta, or of its larger branches, is always a suspicious sign, and ought to lead us to further inquiry; but inasmuch as it may arise from a projecting spicula, or from the pressure of a tumour, it is of no value unless conjoined with circumscribed dulness, and even these are not conclusive unless associated with other confirmatory phenomena.

DELAY OF THE PULSE is a sign of atheroma, or of dilatation of the aorta, but not of saccular aneurism, which does not interfere with the normal propagation of the pulse.

\* *Edinburgh Medical and Surgical Journal*, vol. xlv. p. 316.

† *Vide* Lecture I., p. 31.



A knowledge of the anatomical relations of the thoracic aorta teaches us, that the prevalence of one or other of these symptoms may be accepted as a certain indication of the site of an aneurism, and often of its size. *Pain* on the right side is of little consequence, as an aneurism coming from the ascending aorta, and passing to the right, lies in front of everything, and already implicates the anterior chest wall before pain becomes a prominent symptom. On the left side it is different, and so-called rheumatic pains about the left shoulder ought to be most carefully inquired into, as they will often be found to depend upon an aneurism of the upper part of the descending aorta. In this situation also, if the aneurism is of any size, we frequently have either dilatation or contraction of the pupil; but this is always an inconstant symptom, and never an early one, it is therefore of less importance than pain. *Brassy, spasmodic cough*, and *vox anserina*, lead us at once to the left edge of the sternum about the first interspace, just over the left end of the transverse portion of the arch of the aorta where the left recurrent winds round it. *Dyspnœa*, not accompanied with brassy, cough, or *vox anserina*, and not associated with any obvious affection of the lungs or heart, should lead us to examine the upper part of the sternum with care, as it will not infrequently be found to depend upon pressure in the trachea or right bronchus by an aneurismal bulging of the first half of the transverse portion of the arch. In rare cases this bulging is strictly limited to the posterior part of the vessel, and then a positive diagnosis is quite impossible, yet the signs and symptoms are even in these cases so well marked and peculiar as almost to make the probability certain.\* Aneurisms in the posterior mediastinum present the fewest symptoms of any, and are seldom even surmised until they produce pain by erosion of the vertebræ, or have become large enough to impli-

\* The case of the late Earl St. Maur will at once recur to the reader, and at p. 1123 of the *Ed. Med. Journal* for June 1872, will be found a similar case, in which an equally successful attempt at diagnosis was made.

cate the lung, or to interfere with the circulation through the *azygos*, *hemiazygos*, or *vena cava ascendens*. When an aneurism springs from the anterior portion of the ascending aorta it often passes to the left, and lying in front of the lungs, just above and in close proximity to the heart, it often escapes observation until by pressure it has produced very considerable disease of the lung.

The diagnosis of abdominal aneurism is either very easy or the reverse. During a considerable portion of its course the abdominal aorta can be readily felt, and any alteration of its calibre distinguished by palpation. Should any tumour be perceptible, auscultation will usually reveal a systolic bruit solely; the second sound is never propagated into the abdominal aorta and a diastolic bruit is almost unknown.\* When the tumour comes from the anterior surface of the abdominal aorta, no pain is ever complained of unless it arise very high up in its course, and almost or quite beyond the reach of palpation. Occasionally, however, flatulence and other symptoms of gastric disturbance are present, apparently depending upon interference with the innervation of the stomach. The immobility of a tumour during respiration is not, as was supposed by Dr Henry Kennedy, a proof of its aneurismal character; it only shows that it is fixed by adhesions or otherwise, but does not by any means prove that it is connected with the aorta. When the aneurism, however, arises from the posterior surface of the aorta, and especially if it be beyond the reach of palpation, we can only arrive at the diagnosis of the aneurism *per viam exclusionis*. By far the most distinctive symptom is the presence of severe neuralgic pain, at some times affecting the bowels, at others passing as a girdle pain round the body, or along the spine into the extremities, and otherwise unaccountable; this pain, though tolerably persistent, is subject to variations like all other

\* The only recorded case of a diastolic murmur in abdominal aneurism, that I know of, is one narrated by Dr Wickham Legg in *St Bartholomew's Hospital Reports* for 1880, vol. xxi. p. 258, but I myself have also met with one other similar case.

aneurismal symptoms, and now and then disappears for months at a time; this unaccountable disappearance may, in obscure cases, be regarded as to a certain extent confirmatory of the diagnosis of aneurism. Dr Beatty of Dublin has related a most instructive case of abdominal aneurism, in which the tumour lay between the crura of the diaphragm, and in which the character of the pain led ultimately to a correct diagnosis.\* It is to be regretted that in this case no percussion nor auscultation were instituted down the spine, for there can be little doubt that the nature of this case might have been thus ascertained with a fair amount of precision; and this should never be omitted in any similar instance, as the discovery of a localised dulness or arterial whiz would materially aid the diagnosis of such cases.

The course of an aneurism is usually chronic; it may last for many years, till death takes place, either gradually by asthenia or dropsy, or more suddenly by the occurrence of oedema, gangrene, or inflammation of the lungs, or from rupture of the sac either externally or internally into one of the serous cavities or into a mucous canal. Death by pure asthenia is comparatively rare, a gradually increasing marasmus being usually hastened to its fatal termination by the occurrence of some acute complication or intercurrent disease, or by rupture of the sac. On the other hand, rupture of the sac is not always immediately fatal; rupture into a serous cavity usually is so, but there are many cases of so-called "weeping aneurisms" in which the sac communicates with some mucous surface, and occasionally pours forth small quantities of blood at irregular intervals for months or years. When the communication is with the alimentary tract, this leakage is not so obvious, except when so copious as to give rise to hæmatemesis, a state of matters but rarely followed by recovery, and yet at least one case of this has been recorded.† But when the blood is poured into the respiratory tract, attention is at

\* *Dublin Hospital Reports*, vol. v. p. 166.

† *Gairdner's Clinical Medicine*, Edinburgh, 1862, p. 495.



once directed to this important symptom. In Mr Liston's case the period which elapsed between his first copious hæmoptysis and his fatal hæmorrhage was five months, without any intervening hæmorrhage; and Dr Gairdner has related a remarkable case, in which from the first leakage to the last fatal effusion, no less a period than five years elapsed, during which there were repeated slight attacks of hæmorrhage.\* But the most extraordinary cases of intermittent hæmorrhage are those recorded as having taken place from the external surface. Thus Neligan relates the case of a ship carpenter, with an aneurism of the aorta opening externally about the second rib on the right side in front, which for more than a year discharged blood at intervals; sometimes so copiously and in so full a stream as to be with difficulty arrested. Three weeks after his last hæmorrhage his aneurism underwent a marked abatement, and he left the hospital declaring himself quite well.† Nor is this case unique; several somewhat similar having been recorded. Perhaps the most extraordinary of these is one related to the late Mr Syme, by Mr Ramsay of Broughty-Ferry, of a man affected with aneurism of the arch and of the innominate, which ruptured opposite the cartilage of the third rib; a stream of blood is reported to have issued somewhat thicker than a quill; the patient, nothing alarmed, held a bowl to receive the contents of what he supposed to be a bloody boil, and even squeezed it with his chin to empty it faster; when he had lost about a quart of blood he fainted, and the bleeding stopped. Four months subsequently he died of typhus, no new bleeding having occurred.‡ Thus even rupture of an aneurism, usually and rightly regarded as almost necessarily fatal, does not always make further treatment utterly hopeless; and if this be the case with a ruptured aneurism, with how much more hope ought we not to attempt the cure of one still unruptured. Nature unaided has not

\* Gairdner's *Clinical Medicine*, Edinburgh, 1862, p. 509.

† Stokes' *Diseases of the Heart and Aorta*, Dublin, 1854, p. 582.

‡ *Monthly Medical Journal*, January 1850, p. 89.

infrequently succeeded in promoting the cure of such cases ; and the medical art would surely be unworthy of the confidence reposed in it, were it incapable of following up and improving upon the hints afforded by nature even in this most serious disease. For nature can only cure under certain conditions and in certain circumstances, but art can change the latter, and modify or fulfil the former. Art, therefore, properly directed, ought to cure more than nature ; and as no cases are utterly hopeless, even when left to nature alone, so all ought to be more or less hopeful when under the guidance of art ; and the more hopeful, the more intelligent the art that guides them is. The spontaneous or natural cure of aneurism is effected in three modes :—First, by sphacelus of the tumour, the arterial opening remaining closed ; secondly, by obstruction of the artery to which the tumour is attached ; and thirdly, by gradual occlusion of the tumour with fibrine, the artery to which it is attached remaining pervious.\* The first mode of cure has only been produced as it were accidentally, and cannot be imitated by art. The surgical treatment of external aneurism is based upon the second, which is obviously inap-

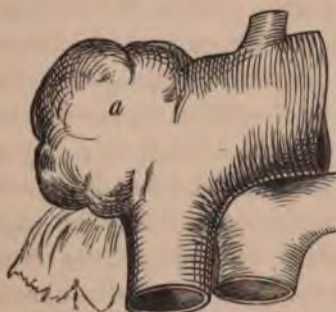


Fig. 24.

applicable to the treatment of large internal aneurisms. The medical treatment of internal aneurisms seemed therefore to be confined to an endeavour to bring about the third method of cure, but I hope to be able to show you that we have a fourth method, strictly scientific in its character, always certain to give relief, and

which, if begun early enough, is sure to prolong life longer than any other method yet devised. Of the spontaneous cure of aneurisms of the aorta, I myself have only seen two

\* *Vide Hodgson On Aneurism*, London, 1815, p. 101. Many cases of the spontaneous cure of aneurisms will be found in this work.



instances. The first was a small aneurism, rather larger than a large walnut, wholly filled with a firm, pale, fibrinous clot, and attached to the upper part of the ascending aorta, just where it passes into the transverse; it was removed from the body of a man who had died from the fatty form of Bright's disease (large white kidney), and who had not at any time presented symptoms of aneurism.\* The second case was that of an aged woman, an inmate of one of our eleemosynary hospitals, under the care of Dr Gillespie. In this case the tumour arose from the descending part of the aorta, just where it passes off from the transverse portion; it passed backwards into the left

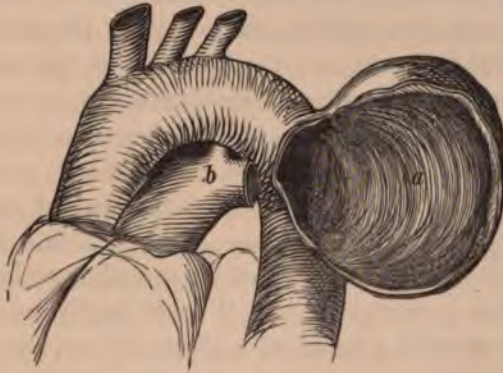


Fig. 25.

vertebral sulcus, and pressed upon the left lung, hæmorrhage from which, unconnected with the artery, but depending upon the disorganisation produced by the tumour, had proved fatal. The aneurism was as large as an orange, was wholly filled with a laminated clot, and had presented none of the usual symptoms; it had been, in fact, wholly unsuspected. In this case the whole of the arch of the aorta was converted into bone, and was as firm as the handle of a walking stick.† Attempts have been made in various ways to produce this

\* *Edinburgh Medical Journal*, July 1870, p. 82.

† Both of these preparations are in the Museum of the Royal College of Surgeons, Edinburgh.



favourable termination. Those depending for their success upon the employment of ordinary sedatives have so signally failed as to require no notice here. Even their use as palliatives has been unproductive of much benefit, and the most powerful anodynes have been almost useless in relieving the excruciating agony which is so frequently an accompaniment of this terrible disease.

Of all the more modern attempts to relieve or cure this affection, there are six which seem worthy of mention, and the first I shall notice only because it is modern, and not because its employment has been either markedly beneficial, or apparently deserving of imitation, but rather because its use in these days affords the strongest possible testimony to the dangerous character of the disease, and the inefficacy of the remedies hitherto employed. I refer to the INTRODUCTION OF FINE IRON WIRE into an aneurism with the view of providing an extensive surface upon which the fibrine might coagulate. Twenty-six yards of such wire were introduced by Dr Murchison and Mr Charles H. Moore, through a fine pointed canula into a saccular aneurism of the ascending aorta. The treatment was unsuccessful, and it could hardly have been otherwise in the case referred to, as it was not employed until it was clear that the man had not many days to live. Why it was resorted to in such a case is not very obvious. The proposers of this treatment contend that their experiment showed that the principle upon which it was based was sound, and that further experiments are justifiable.\* But surely nothing could justify so hazardous an experiment except the full conviction that no treatment less dangerous was available; and to this I of course demur, as I think I shall have occasion to show that we have a treatment free from danger, the success of which completely precludes our recourse to any similar experiments. Besides, this treatment is of course only applicable to saccular aneurisms, and for these when distinctly recognised, even though internal, we have a much less

\* *Medico-Chirurgical Transactions*, vol. xlvii. p. 129. London, 1864.

dangerous and probably a more efficient curative agency in ELECTROLYSIS. Unfortunately, in regard to internal aneurisms, we can never be thoroughly certain that we have to do only with a truly saccular aneurism, and in any other the risk of embolism, though greatly exaggerated, is still so great that the employment of galvano-puncture in aneurism is chiefly restricted to those cases of arterio-venous tumours termed "cirroid aneurism," in the treatment of which its use is most successful, but only so long as the arterial element prevails; whenever the venous element is in excess I can say with perfect certainty that it is of no use, if the disease is of any extent. In truly medical aneurisms, it is only available for the prevention of external rupture, for which—failing other means—it is admirably adapted, and therefore deserving of a short notice. Originally suggested by Pravaz for the cure of external aneurisms, in the treatment of which it has had a fair measure of success, as may be seen by a reference to the tabular statement given in Ciniselli's memoir,\* to which I need not now further refer. Its employment has been extended to thoracic and abdominal aneurisms by Ciniselli,† by Drs James and John Duncan,‡ and by Dr Decristoforis;§ these include eight cases of thoracic aneurism, in only one of which was any amount of success attained;|| the others were all unsuccessful; and one case of abdominal aneurism in which complete success seemed to be attained, but in which premature exertion was followed by the sudden death of the patient.¶ The results attained, therefore, in the treatment of internal aneurisms, have not been such as to induce us to hope much from galvano-puncture as a remedial agent in this

\* *Sulla Elettro-Puntura nella Cura degli Aneurismi*, Cremona, 1856; and *Edinburgh Medical Journal*, April 1866, p. 926.

† *Op. cit.*

‡ *Edinburgh Medical Journal*, April 1866, p. 920; and August 1867, p. 101.

§ *Aneurism dell' Aorta Ascendente Trattato colle Elettro-Puntura, Caso Clinico*, Milan, 1870, &c.; and *Edinburgh Medical Journal*, December 1870, p. 537.

|| *Loc. cit.* p. 540.

¶ *Edinburgh Medical Journal*, p. 922.

class of disease, while the unavoidable risks of embolism are so great as to lead to its abandonment by almost all as a curative agent in such cases. On the other hand, the ease and rapidity with which clots can be so formed, is such as to most certainly lead to its employment as a *dernier ressort* in all cases where an external rupture is imminent. For this purpose steel needles are employed, about 5 inches in length, and of the thickness of 0.07 inch, or No. 16 of wire-gauge; of this the non-insulated portion measures  $1\frac{1}{8}$  inch in length, while the insulated portion, or that covered with vulcanite (or ebonite), measure  $3\frac{3}{8}$  inches in length. If the steel be not gilded, the positive pole is rapidly corroded; but when the wire is of the thickness described, it is not acted on to such an extent as to have its efficiency seriously impaired; besides, when blood is the electrolyte, its corrosion results in the production of ferrous chloride, and, probably, of ferrous sulphate, salts which coagulate albumen and thereby increase the wished-for result.\* Four cells of a Bunsen's battery are effectual, and produce no pain, or only a bearable amount of it; but as many as six cells sometimes produce so much pain as to render the patient uncontrollable:† so that if it should be thought desirable to use more than four cells, the patient ought to be placed under chloroform.

The battery should have a continuous current, and the needles should be introduced from the same side, near the base of the external tumour, parallel to each other, and one or two inches apart. The number of the needles employed may be multiplied, if the aneurism be large, and the action may be prolonged till pulsation ceases, or till gas can be clearly detected on percussion. A *séance* of about twenty minutes is usually enough for one application, and this may be repeated as necessary, according to the circumstances of the case. Such is a succinct account of the method of employing galvanopuncture in aneurism,—a method of treatment not, however,

\* *Edinburgh Medical Journal*, August 1867, p. 119.

† *Loc. cit.* p. 109.



applicable to the cure of internal aneurisms, on account of the risks connected with it, but one which may certainly be advantageously employed as a means of prolonging life in exceptional circumstances.

Last winter (1874-5) we had a very instructive example of the use, or rather of the inutility, of galvano-puncture in the cure of aneurism.

CASE XXXIII.—Alexander M'Intosh, a mason, aged 34, residing at Newport, Fife, admitted on 12th October 1874 to bed 10, Ward V., complaining of a pulsating tumour at the root of the neck, and another in the region of—and actually beneath—the right shoulder-blade. He is a hardworking man of temperate habits, with a comfortable house, and good family history, who had for a long time met with no more serious accident than a fall on his right elbow joint about six months before admission. About six weeks before admission he was, while at work, seized with a pain between his shoulders; it was in itself of no consequence, and did not prevent his working, but it attracted his attention to the spot. He discovered a swelling, which was as large then as on admission, and on consulting his doctor he was told to stop work and was sent into the Royal Infirmary. He has now no pain, only a stiffness over the right shoulder and upper part of the chest. With the exception of an attack of fever, about ten years ago, which laid him up for two months, his health has always been good. He is a well-developed man in good condition, weight 11 st. 4 lb., height 5 ft. 7 in., skin natural, temperature 98·4. For the last six weeks he has suffered occasionally from palpitation, and has a feeling of stiffness about the upper part of his chest in front. Both radial pulses are synchronous and of nearly equal force, though at times the right seems the feebler, pulse weak, 90 per minute; above the right clavicle there is a lobulated bluish swelling, which commences a little to the right of the middle line and runs round the root of the neck to the back, about the middle of the clavicle it measures 3 inches across. Posteriorly in the region of the right scapula there

is a visible pulsation, the margins of which are ill-defined; beneath, the pulsation extends a little beyond the scapular angle; internally, where it is most prominent, it almost reaches the middle line; externally, it reaches the posterior fold of the axilla; and above, it rises to the level of the vertebra prominens, and merges into the swelling at the root of the neck. The colour of the posterior pulsating tumour does not differ from that of the natural skin. On palpation the swelling over the clavicle feels soft, fluid, and relaxed, except at one spot just above the clavicle, where a firm and more elastic tumour is to be felt. On placing the hand lightly over this soft swelling a purring tremor is felt, but no distinct pulsation until we press deep enough to reach the elastic tumour just referred to, which feels rounded and beats a little later than the apex of the heart. The pulsation posteriorly is heaving, and lifts the scapula at each beat; at its upper part there is to be felt a purring thrill similar to, but less intense than that in front. Firm pressure on the elastic tumour referred to as projecting above the clavicle stopped the thrill both in front and behind, and greatly diminished the size of the tumour behind as well as the force of its pulsation. The cardiac apex beats in the fifth interspace, 3 inches from the middle line; the cardiac pulsation seems to occupy a larger space than usual, but is not forcible, there is no visible pulsation in the episternal notch, but there is considerable heaving over the upper part of the right side of the chest. The cardiac dulness does not rise above the third rib; at the level of the fourth costal cartilage it commences at the right margin of the sternum and extends across for a distance of 5 inches. On the right side anteriorly the upper part of the chest is dull down to the third rib; as low as the first interspace this dulness reaches the middle line, beneath that there is a space of about 2 inches to the right of the middle line where the percussion note is more resonant than over any other part of the chest. Posteriorly the whole right half of the chest is dull down to the lower angle of the scapula, beneath this there is about 2 inches of normal resonance

gradually shading off into complete liver dulness. The percussion of the lungs on the left side is normal both in front and behind.

On auscultating over the apex the first sound is found to be impure, the second unusually loud. Over the upper part of the sternum and the dull part to its right, we have a rough double murmur audible as low down as the fourth costal cartilage; here, however, we have only a systolic murmur followed by a second sound. In the pulmonary area the second sound is obscured by the diastolic murmur, but may be picked up by passing the stethoscope a little to the left in the same plane. The rough double murmur continues audible over the innominate, but over the rounded pulsating tumour above the clavicle only a loud systolic murmur is audible. Over the bluish lobulated venous tumour, especially close to the trachea, a loud purring whiz is heard, which is propagated so loudly into both carotids as to obscure any other acoustic phenomena which might be audible in them. Over the upper part of the posterior tumour there is a loud whiz, over its lower part merely a systolic bruit. Over the left lung the respiration is vesicular, but somewhat feeble. On the right side the respiration is obscured by the murmurs described, except in the part beneath the scapula, where the respiration is vesicular but slightly higher in pitch than on the left side, and the same remark may be made as to the respiration on the lower part of the right lung anteriorly. His other organs and systems are normal.

Here we had apparently to do with an aneurism of the aorta, involving the innominate and the subclavian, with a bulging projection from the latter vessel which communicated with the veins of the neck, its history and mode of origin being obscure. It was impossible to decide, however, what the posterior pulsating tumour actually was. The patient was seen, and carefully examined by almost all of the physicians and surgeons attached to the Infirmary, but no definite opinion could be arrived at. It seemed to be either an aneurismal



dilatation of the posterior scapular artery, or a cirroid aneurism involving it and its branches; there was no reason to suppose that it arose from the aorta perforating the chest posteriorly. In either of the two cases supposed, it seemed to be a most favourable case for galvano-puncture, all the more that the pulsation could be almost completely commanded by pressure above the clavicle. My colleague, Dr John Duncan, whose large experience in regard to galvano-puncture is so well known, was kind enough to undertake the treatment of the case under my supervision, and continued it for more than six months. After each application of the needles the circulation in the tumour was commanded, and the pulsation of the tumour suppressed by a large bag of shot firmly bandaged over the supra-clavicular space. After each application the usual results followed, the development of a certain amount of gas and the consequent diminution of the pulsation, but it was several months before any firm nodular coagula could be detected; at last, however, these made their appearance and gradually spread, till we hoped that a cure of this part of the poor man's ailment was about to be secured. This hope was however delusive, the coagula never accumulated and coalesced; on the contrary, what seemed to be gained at one *séance* was too often lost before the next, though they were not unduly postponed, but repeated as frequently as possible; at last he was discharged on 8th May somewhat improved, and the pulsation beneath the scapula somewhat lessened. I have, however, since that time seen him at his own house in Newport, and found him very much as he was upon admission, all that seemed to be gained having been entirely lost.\* You will see then that this case fully bears out all that I have said in regard to this method of treatment, and that even in apparently the most favourable cases it is not much to be depended upon.

Comparatively recently the HYPODERMIC INJECTION OF ERGOTINE has been recommended by Professor Lagenbeck of

\* This patient still survives in much the same condition. Since his discharge he has become the father of three daughters.

Berlin as a curative agent in the treatment of aneurism, on the strength of two cases which were thus successfully treated by him. The first was an aneurism of the subclavian and innominate, in a man aged forty-five, a pulsatile swelling the size of a closed fist existing in the supraclavicular fossa of the right side, accompanied by excruciating pain in the right arm, and consequent sleeplessness. From the 6th of January to the 17th February 1869, injections of ergotine, varying from half a grain to three grains, were made at regular intervals of about three days; and this treatment was followed by great improvement in the patient and considerable diminution of the tumour. The second case was a saccular aneurism of the radial artery, in a man aged forty-two. One single injection of ergotine caused complete subsidence of the tumour. Next day it returned somewhat, and slight swelling and infiltration could be felt in the neighbourhood of the puncture. By the end of eight days these had all disappeared, and not a trace of the tumour could be perceived.\* Langenbeck is too well known as a careful observer to permit these cases to be ignored; at the same time, if the action of ergotine be, as he supposes, on the unstriped muscular fibre, it is difficult to perceive how it could possibly have any effect in this manner on the larger tumour described, as the muscular fibres surrounding it must have been few and sparse indeed. It is possible that this treatment may have some effect on the small aneurisms of the smaller arteries; but it seems impossible, for the reason stated, that it can have any effect on large aneurisms, especially of the larger arteries. I have employed it repeatedly, and in one case continuously for many months, without observing the very slightest result, and this though the preparation was unquestionably active, as evidenced by the very excellent results I obtained from its use in hæmorrhages both in hæmoptysis and in epistaxis. It would, however, be unfair to omit allusion to this treatment in any treatise

\* *Berliner Klinische Wochenschrift*, March 1869; and *Edinburgh Medical Journal*, November 1869, p 461.

purporting to present modern views of the treatment of aneurism. The preparation employed by Langenbeck was Bonjean's watery extract, diluted with three times as much of rectified spirit and of glycerine; that which I employed was prepared in this country, and was undoubtedly active, both as proved therapeutically and also physiologically, one grain of the preparation hypodermically injected sufficing to stop completely the circulation in a frog for fully five minutes.\*

PRESSURE, as a mode of treating aneurism, is of course wholly inapplicable to thoracic aneurism, and also, as a rule, to abdominal aneurisms. One case of abdominal aneurism has, however, been recorded by Dr William Murray of Newcastle-on-Tyne, in which pressure of the aorta immediately above the tumour was successful in curing the disease. The first attempt in this case, a man aged twenty-six, failed; on the second attempt the patient was kept for five hours under chloroform, and pressure was fully maintained by a properly constructed tourniquet. The result was that in three months the patient was at his work as an engine-fitter, perfectly cured; the aorta beneath the tumour, the iliac, and the femoral arteries, being quite pulseless.† Cases in which this mode of treatment is available must certainly be very rare, but it is proper to bear it in mind as a remedy capable, in fitting cases, of producing a much to be desired result in a very short time.

The treatment which has, however, for long been deemed the most suitable for internal aneurisms is that denominated, from its strongest advocate, VALSALVA'S TREATMENT, though, as we learn from Morgagni, Albertini had an almost equal predilection for its use. This, when pushed as far as its originator desired, consisted in weakening the patient by

\* *Vide* a paper by the late Dr Hirschfeld, on The Action of Ergot and Ergotine, in the *Medical Press and Circular*, February 1870, p. 139.

† *Medico-Chirurgical Transactions*, vol. xlvii. p. 187, London, 1864; and *Medical Times and Gazette*, April 1865, p. 383, in which is a further report of the case, showing the cure to have been complete. *Vide* also *The Rapid Cure of Aneurism* by William Murray, M.D., M.R.C.P. Lond.: J. & A. Churchill, 1871.



repeated bloodlettings, and by gradually diminishing his meat and drink till only half a pound of pudding was taken morning and evening, with only a measured quantity of water;\* so that at last the patient was so much reduced that he could not lift his hand from the bed in which, by Valsalva's orders, he lay from the commencement of the treatment. After this the quantity of nutriment was gradually increased, till the patient's strength was restored. That this treatment has been useful in certain cases we have the testimony of many physicians, and it can hardly be thought necessary now-a-days to adduce *seriatim* evidence in favour of a practice originally suggested by Hippocrates,† and which after a varying fashion has kept its place in therapeutics ever since.‡ In recent times this practice was attempted to be revived in Edinburgh by the late Dr Bennett, who expressed his determination to treat his next case more vigorously; because a former patient, after forty days' treatment, during which he had been repeatedly purged freely, twice had a dozen leeches over the tumour, was once bled to ʒxij, and several times to syncope, besides being kept on extremely low diet, including ʒij of steak and ʒij of bread for dinner, yet retained sufficient bodily strength to walk two hundred and fifty yards to the nearest cab-stand on his way from the city.§ But, in spite of its occasional success, modern physicians have almost unanimously agreed to discard it, and, as we have seen, have adopted the most hazardous procedures rather than have recourse to it. And this arises from two reasons, the first of which, and that really the least important, is the very great difficulty with which it can be carried out. In hospital patients it is all but impossible to do so efficiently, on account of the childish selfwill of the patients, and the facilities they have for transgressing; it is only among the better classes

\* Morgagni, *De Sedibus et Causis Morborum*, Letter xvii. Art. 30.

† *De Morbis*, lib. i., n. 10. So Hodgson says, the reference and the fact seem both doubtful; possibly he may found upon the 6th Aphorism.

‡ *Vide* Hodgson, *op. cit.* p. 145, &c.

§ *Monthly Medical Journal*, 1850, p. 169.

## LECTURE XVII.

## ON THE TREATMENT OF ANEURISM BY IODIDE OF POTASSIUM.

GENTLEMEN,—Of all the various modes of treating internal aneurism, there is not one hitherto mentioned which is not attended with considerable risk or danger except Mr Tufnell's plan of perfect rest, while the advantages to be derived from some of them are, to say the least, very problematical. But the treatment by IODIDE OF POTASSIUM, of which I shall now speak, is perfectly safe and free from all risk, and it is equally certain to afford relief, at least I have not yet seen any case where relief was not attained, though naturally enough that relief is not always to be got instantaneously, but requires the treatment to be continued for some time. It also relieves the pain and all the other symptoms of aneurism more rapidly and more effectually than any other treatment, apart even from the powerful agency of the recumbent posture; and for the time it has been in use it has given greater and more permanent relief to a larger number of cases of aneurism than any other mode of treatment whatever. Indeed, the relief to the pain and other symptoms is so great and so speedily obtained, usually from the action of the drug alone, that it is often difficult to get the patient to submit to any restrictions. Besides, it is not always necessary. I was acquainted with a gentleman who in 1868 had a very large and forcibly pulsating aneurism arising from the ascending aorta and passing to the right side, who was permitted to go about in moderation

during the whole period of his treatment, and in whom, nevertheless, the larger portion of the sac became consolidated, so that in 1871 he had only a comparatively feeble pulsation in its upper part. This gentleman continued the treatment steadily for an entire year, and continued to take the iodide at intervals. He was so well that for two autumns he shot regularly on the moors. He was so active that it was almost impossible to restrain his movements, yet he received so much benefit that there is every probability that the adjuvance of so powerful a remedy as the recumbent posture would in him have promoted an almost perfect cure. His unrestrainable activity at length cut him off, for after a convalescence of five years he insisted on going salmon fishing, and stood for a whole forenoon up to his waist in water on a cold autumn day, wielding a heavy salmon rod. The natural result occurred, he had an enlargement of his aneurism, a series of fainting fits, and a serious illness, from which he at length recovered not much the worse apparently. Hardly convalescent he went off to business, caught a fresh cold, which confined him to bed, and about a week afterwards he ruptured the aneurism in a fit of coughing, and died.

I need hardly say that aortic aneurism, even of considerable size, is not always incompatible with an active life. A few years ago there was said to be in Dublin a prize-fighter by profession, who had a large aneurism projecting through his sternum; yet he suffered so little that he continued to maintain himself by his repulsive calling, only taking the precaution to put an iron cage over the tumour when he went to battle. And one of the cases subsequently related, for ten years maintained himself as an hotel porter, with a large aneurism projecting through the walls of his chest. So that mere continued immunity from suffering, and capacity for exertion, prove nothing. But when we find a drug relieving suffering, mitigating every other symptom, and restoring capacity for exertion when lost, and that not occasionally, but in almost every case in which it has been fully tried, all who



know anything of aneurism will agree with me that it must be a remedy deserving of the fullest confidence. Further, when we find, as we do find, that this remedy is capable of producing these favourable results apart from any adjuvance of diet or posture, our confidence in the truly remedial character of the action of the drug must surely be confirmed, especially when we find its curative action to be founded on a strictly physiological basis.

As has been so often the case in medicine, the adoption of this mode of treatment has not been the result of any speculative ideas as to the nature of the particular disease, or the actions of the special remedy, but has been entirely based upon empirical observations—observations so opposed to all our preconceived ideas on both of these points, that for long it was impossible to give any intelligible explanation of them. The late Dr Graves of Dublin, *facile princeps* of all our clinical writers, was the first to direct attention to the beneficial action of large doses of iodide of potassium in painful rheumatic affections of the fasciæ and nerves; and it is now many years since the late Mr Craig, formerly of Ratho, informed me that a patient of his, while being treated with that remedy in dozes of seven grains thrice a-day, for concomitant rheumatism, had obtained complete relief from the neuralgic pains arising from a large aortic aneurism. It struck me at the time that the unexpected relief was perhaps not accidental, and that the treatment was worthy of further trial. No opportunity, however, occurred to me of putting it to the test.\* Meanwhile, about 1859, a Pole presented himself to M. Nelaton, with a tumour in the lower part of the neck, which had been recognised by MM. Bouillaud, Andral, and Beau as an aneurism of the innominate implicating the aorta. He stated that while in Warsaw his sufferings had been much relieved by the use of the iodide of potassium.

\* I may say, however, that I had already treated, and was about to publish, my three first cases, XXXVI., XXXVII., and XXXVIII., before I became acquainted with the history of the treatment about to be narrated.

M. Nelaton took the hint, and continued the remedy, the result being, to his great surprise, a notable amelioration of all the symptoms, with almost complete disappearance of the tumour, so that the Pole returned home in a satisfactory state of health. M. Bouillaud was the next to follow out this plan of treatment in four cases which he has related. One was a female, with an aneurism of the carotid the size of a pigeon's egg; she had fifteen grains of the iodide of potassium for several days, and afterwards thirty grains a-day for two months. At the end of this period the tumour had diminished so much that it might be considered to have completely disappeared. The second case was an aneurism of the brachiocephalic trunk and aorta in a male, which, under the same treatment, underwent considerable displacement and well-marked diminution in size. He was still under treatment when these cases were published. The third case was one of a large aneurism at the point of origin of the carotid and subclavian in a male, which was considerably diminished in size in a few weeks, under the same treatment; and the fourth case was a carotid aneurism in a man, which had also almost completely disappeared in a few weeks, under the same treatment.\*

It is somewhat singular that, while this problem was thus being experimentally worked out in our Western hemisphere, an independent observer in the Eastern one, having had his attention casually directed to this peculiar action of iodide of potassium in aneurism, was also empirically investigating the subject with no less remarkable results. On commencing hospital duty in August 1860, Dr Chuckerbutty † of Calcutta found in the wards an Irishman, aged fifty, afflicted with a harassing cough, accompanied by profuse expectoration which remained unrelieved by any treatment until a solution

\* These cases are referred to in Dr Roberts' paper, presently to be quoted; they are detailed in the *Clinique Européenne* for July and August 1859. M. Bouillaud's cases are also quoted by the *Union Médicale* of 8th March 1859, as having been contained in a clinical lecture published in the *Gazette des Hôpitaux* of the same year.

† *British Medical Journal*, 19th and 26th July 1862.

of iodide of potassium, in decoction of cinchona, was employed. Coincident with the relief to the cough thus obtained, Dr Chuckerbutty was astonished to find that an aneurism of the innominate, from which this man also suffered, became gradually solidified. This aneurism projected above the sternal notch, and was at first the size of a walnut, with thin walls, and readily emptied; it grew steadily upwards into the neck, passing beyond the median line, till it attained the size of an orange, subsequently becoming hard and consolidated under the use of the remedy referred to. Some months afterwards the man died of an attack of bronchitis, and the aneurismal sac was found to be as large as a pear, filled with dense coagula, which left merely a narrow channel on their outer aspect, through which the right carotid and subclavian communicated with the aorta. Suspecting that there might be some causal connection between the remedy employed and the curative result so unexpectedly attained, Dr Chuckerbutty proceeded to treat several other cases of aneurism in the same manner, and of these he has related three. In the first of them, a man, aged forty-seven, with an immense aneurism of the aorta, great temporary relief was obtained from the use of four grains of the iodide of potassium thrice a day. This treatment was continued from 7th December to 12th January when death occurred from rupture. Three hours after death the sac was found filled with dense solid coagula. In the second case, also a large aortic aneurism in a male, though under treatment for less than three weeks, there was so great a temporary amendment, that the man thought himself cured, and insisted on leaving the hospital to spend Christmas with his friends. He did not return till after the lapse of more than three months, and then his symptoms were all intensely aggravated, and he died in three days. The third case was that of a man with an aortic aneurism, projecting as a dome-shaped tumour, two inches in diameter, through the sternum. Various remedies had been previously ineffectually employed in this case, but, under the use of iodide of potassium inter-



nally, along with the application of tincture of iodine externally over the tumour, the patient steadily improved, the thoracic pain disappeared, the swelling diminished, the hæmoptysis ceased, and the patient ate and slept like any other man. He was still under treatment when the paper was sent home for publication. Dr Chuckerbutty points out that the consolidation of the contents of the aneurismal sac is the important fact in the histories of these cases, and contends that this probably depends upon some hitherto unsuspected action of the iodide of potassium on the blood; and this opinion seems also to be shared by Dr Roberts of Manchester, to whose interesting paper I shall now refer.

About the beginning of 1862, Dr Roberts was consulted by Mr T. Windsor in regard to a case of aneurism, and his attention for the first time directed to the important remedial action of iodide of potassium in that disease. The case was that of a lady, aged twenty-nine, who had an aneurism of the aorta, implicating the origin of the innominate; there was excessive pulsation at the lower part of the neck on the right side, repeated slight attacks of hæmoptysis, occasional loss of voice, dysphagia, constant troublesome cough, with scanty expectoration, and recurring paroxysms of pain and dyspnoea so severe as to compel her to get up and walk about. Under a regulated diet and strict confinement to the recumbent posture, her condition steadily became aggravated; she became so weak and emaciated as to be unable to leave her room, and the right clavicle began to project half an inch beyond its natural level, while the pulsation was much increased. In April 1863 she was put upon iodide of potassium, with the view of relieving the severity of the pain, and this object having been attained, the remedy was discontinued, after having been employed for only one week. At last, in July 1862, when she was so much worse that death seemed not far off, Mr Windsor put her upon five grains of the iodide of potassium thrice a-day, subsequently increased to ten grains, and again reduced to five on account of the occurrence of

violent salivation. This latter dose agreed, and the patient continued taking it up to the time of the publication of the case,\* the result of the treatment being a rapid subsidence of all the general symptoms, complete cessation of the cough, pain, dysphagia, and hæmoptysis, while the patient gained flesh and strength, and, in a few months was able to walk six miles, the clavicle having subsided to its normal position. Dr Roberts† subsequently treated in the Manchester Royal Infirmary a male patient, aged thirty-nine, in whom the first bone of the sternum and its vicinity were the seat of heaving pulsations, while in the second left intercostal space there existed a conical, soft, pulsating elevation, projecting about a quarter of an inch, and with a base the size of a shilling. Over the bulging parts there was an area of dulness, measuring transversely four inches and a half, and vertically three inches and a half. There was pain in the left side of the head and shoulder, dyspnœa, dysphagia, and some cough. The patient was put upon a restricted allowance of fluids, and had five grains of iodide of potassium given him thrice a-day. In three days the dose was raised to seven and a half grains thrice a-day; and in six days the patient expressed himself as much better, his pain gone, and the cough and difficulty of breathing less troublesome. The dose of the iodide was then raised to ten grains thrice a-day, and after seventeen days' treatment, the pain, dysphagia, and dyspnœa were quite gone; the soft pulsating tumour had entirely subsided; he was allowed to get up, and his restrictions were relaxed. The dose was now increased to fifteen grains, and after twenty-five days' treatment, to twenty grains, thrice a-day; but after this dose had been continued for about five days, it had again to be reduced to five grains, on account of the supervention of diarrhœa. The bulging was decidedly less, the area of dulness reduced to three inches and a half transversely, by two inches and a half vertically; the elevated tumour had quite disappeared, and the general pulsation was almost gone, but

\* *Vide British Medical Journal* for 24th January 1863.

† *Loc. cit.*

the enlarged superficial veins, and the stridulous voice, still remained to show that the cure was not complete. In this case decided emaciation was the concomitant, if not the result, of the treatment. Dr Roberts\* also relates a case which occurred under the care of his colleague, Dr Wilkinson, of a large thoracic aneurism in a male, projecting in the neck as a tumour the size of a child's head, extending from the left clavicle to the angle of the jaw, and apparently about to burst. Fifteen grains of the iodide of potassium were administered thrice a-day, the pain was lessened, and the growth seemed stayed, but the patient died in seventeen days from gangrene of the lung, the effect of pressure upon the left pneumogastric nerve. The sac was found lined with coagula, and a large firm blanched clot, attached by a broad pedicle to its upper part, floated free within it.

I myself have employed this method of treatment during the last eight years in a very considerable number of cases, with unvarying success so far as the relief to symptoms is concerned, and with such favourable results as to retarding the further progress of the case, and even in some cases promoting an apparent cure, as certainly stamp this treatment as one of the most efficient hitherto propounded for the relief of this intractable complaint; while it has certainly the not despicable property of being uninjurious to the patient in any respect. I shall give a selection from the cases treated by myself, bringing them down to the present date, so far as known.

CASE XXXVI.—Peter Rice, a mason, aged 39, admitted into Ward III. on April 29, 1867.† Patient has never had rheumatic fever, but for the last five years has been subject to rheumatic pains in his hip, leg, and back, which are generally severe, and are most apt to recur in the changeable weather of spring. He has been in the habit of drinking pretty freely.

\* *Vide British Medical Journal* for 24th January 1863.

† This case was primarily under the care of Dr Warburton Begbie, acting for Professor Laycock, and was transferred to my care when the clinical wards were closed, at the end of the summer session of 1867. The case is partly condensed from the Report in the clinical records of Ward III.



About twelve months before admission he fancied he was overwrought, and when he got home, he found he could not take a deep inspiration freely; he also felt a sharp pain at a point about two inches to the right of and a little above the left nipple. This pain has continued ever since, and has latterly increased considerably. It is stationary at the part described, but when more severe than usual, it spreads upwards to the arm-pit and shoulder, and down the left arm to the wrist, occasionally extending downward to the scrobiculus cordis, and sometimes striking sharply through to a corresponding point in the back. It is specially apt to be severe at night, preventing him from sleeping, and is very annoying when it affects his back, as he cannot lie in any other position than supine. He has occasional fits of dyspnoea, and it is always painful for him to take a full inspiration. He has also some difficulty of swallowing. He is much troubled with palpitation and a distressing feeling of pulsation, and these sensations annoy him most when the pain is severe. At such times he obtains some relief by relaxing the respiratory muscles by stooping forward and leaning against a wall or other support, with his hands and arms extended. About a week ago his symptoms had increased so much that he was obliged to cease working altogether. His appetite is good; his bowels generally confined. On percussion, the heart seems of normal size; its pulsations are distinct, and in their ordinary situation; the first sound is normal, the second accentuated. The right radial pulse is fuller than the left. About three years ago the patient had muscæ volitantes, lasting off and on for about two years, appearing only for a few seconds each time; and still at times he cannot see things at a distance so well as he thinks he ought, and the letters seem to swim before him when reading. His left pupil is slightly dilated. Some time ago he also had tinnitus aurium. His left cheek is often flushed, and at times he feels it warmer than the other. His lung sounds are normal, but he has an imperfect, hard, clinking cough, without expectoration. He also frequently perspires without

any apparent cause. There is a slight bulging of the walls of the chest, between the second and third ribs, at the left edge of the sternum, extending into the manubrium sterni, and gradually declining all round within an area equal to that of the mouth of a tumbler. Over this space there is dulness on percussion, and a distinct sense of liquid pulsation. Within this region the heart-sounds are also extremely distinct, but there is no bruit.

Twenty grains of iodide of potassium were ordered to be given three times a-day, and a belladonna and opium plaster was applied over the tumour. About a month after, on the 23d of May, as the patient fancied he was not improving, the iodide was omitted, and a precisely similar dose of the bromide of potassium was substituted for it. At first the patient fancied the change of remedy had done him good, for he had less pain, and got more sleep; but this improvement was apparently of short duration, even though the dose of the bromide was subsequently increased to thirty grains thrice a-day, for on the 7th of June the iodide was again recurred to in doses of twenty grains thrice a-day, with the addition of one-twelfth of a grain of iodine in each dose. On the 17th of June, it is stated that "there is more pain over the aneurism, and he feels his left hand benumbed. The swelling seems to have increased in size since the 15th instant." On the 7th of July, however, it is entered that he "states that the pain in his breast and down his arm are not nearly so bad as they used to be, so long as he is quiet in bed; but when he rises and walks about, they become even worse than before." The patient continued to wear the belladonna plaster, but on the 7th of July the iodide was omitted, probably on account of coryza, which, though not recorded, the patient told us he suffered from about this time; and on the 8th the following pills were prescribed:—

R Extr. aloës aquosi, gr. j.

„ colchici acetic, gr. j.

Masa. pil. hydrarg. subchlor., gr. ij. Misco.

Fiat. Pil. Mitte tales xij. Sgr., One night and morning.

There is no further record in the books of Ward III. as to the iodide being again resumed, and I am not aware whether it was or not; I believe, however, that it was. The last entry is on 18th July—"Thinks himself easier to-day."

On the 1st of August the patient was transferred to Ward VII., and placed under my care. He was at once placed upon thirty-grain doses of the iodide of potassium three times a-day, and these doses he continued to take without intermission up to May 1868, with continually increasing benefit, and without the production at any time of the slightest unpleasant symptom. For several months he also continued to wear a simple belladonna plaster over the tumour, but at last it blistered him, and produced so much eczematous eruption each time it was attempted to re-apply it, that it had to be discontinued. On coming under my charge he was strictly confined to bed, and for long he lay entirely on his back, that being the only position in which he found always comparative, and latterly, perfect ease. He was only allowed to get up and move about for the first time about three weeks before his dismissal, on the 2d of April. He was placed upon fish diet at first, meat being subsequently given when he tired of the fish, but he was at all times carefully warned of the necessity for strict moderation both in eating and drinking; water, small quantities of tea, and milk for supper, being the fluids supplied. With all this care, and notwithstanding the large doses of the iodide administered, his progress at first was extremely slow, but it was steady. In a clinical lecture given on his case on the 19th of November 1867, I find it stated, "there is no longer any tumour visible, and it is only on careful examination that you will discover any pulsation; he is so far recovered that he is a little inclined to be rash, and to move about quickly in bed, or even to turn upon his side; but upon this the pulsation instantly returns—a sufficient warning to him that he is not yet cured." Indeed, it was not till the beginning of March 1868 that he was able to move about freely without discomfort, or any return of the pain or pulsation.



On the 1st of April 1868 I had the pleasure of exhibiting this patient before the Edinburgh Medico-Chirurgical Society, just previous to his discharge from hospital, when its members had an opportunity of observing the complete subsidence of the tumour described, pulsation being only obscurely felt in the situation where it formerly existed; they also heard the man's statement that his pain, dyspnoea, and dysphagia were gone; and they were able, from his healthy and energetic appearance, to form some idea of the importance and value of this mode of treatment.

A year afterwards, 1869, I reported to the Edinburgh Medico-Chirurgical Society as follows:—Peter Rice, labouring under aneurism of the aorta, has been repeatedly under observation during the past twelve months. When first dismissed he acted as a night-watchman for a month or two; he was then appointed to an institution for the care of orphan children, and he walked about with them, and took them to and from school; latterly he set up a small shop as the easiest means of making his living. He continued to take the iodide of potassium, latterly more irregularly; he continued quite as well, *quoad* the aneurism, as when dismissed; there was the same dull thud as formerly in the second intercostal space, but no pain, nor was any uneasiness ever complained of.\*

Rice continued to present himself occasionally, till at last, after a longer interval than usual, I found him in his working clothes busy laying pavement on the street. He continued to employ himself at his old trade of a mason for two years, and his ability to pursue this laborious occupation for so long and without suffering, is sufficient proof that the relief obtained was not illusive, but a real and decided amendment. And surely it is something akin to a cure to be able to restore to so much usefulness one who so short a time previously had been crippled by the agonies of so terrible a complaint as aortic aneurism. At last Rice died suddenly in the summer of 1872, while working in the Botanic Garden here; he caught a bad

\* *Edinburgh Medical Journal*, July 1869, p. 47.

cold some weeks previously, and, thinking and feeling himself quite well otherwise, he continued at his work as a labourer up to the moment of his death, the immediate cause of which was hæmorrhage into the pericardium, from a very minute rent at a comparatively thin part of the aneurism just where it sprang from the aorta. The aneurism itself was the size of a small cocoa-nut, everywhere dense and firm walled, except just where the rupture took place; it contained only *post-mortem* clots.\*

My next case, though more obscure in its diagnosis, was even more immediately satisfactory in its results.

CASE XXXVII.—John Kerr, a seaman, aged 26, admitted into Ward VII. on October 22, 1867. He states that he has been ill for eighteen months, dating his illness from the privations to which he had been exposed when on an Arctic voyage, and the strenuous exertions he was, while in an enfeebled condition, obliged to make in hauling the boats over the ice and frozen snow. While in America he has been somewhat roughly handled for various diseases with which he was supposed to be afflicted. At last the diagnosis culminated in that of aneurism of the abdominal aorta, and to get relief from this he crossed the Atlantic, and presented himself at the surgical wards of the Royal Infirmary here, and from these he was transferred to my care. He complained of intense pain in the *scrobiculus cordis* extending through to the back, and passing round both sides. On examination, a tumour could be obscurely felt a little below the sternum, and just under the edge of the right ribs; this was more distinctly perceived, falling, as it were, into the hand on turning the patient over on his left side; and in the situation referred to a loud bruit was to be heard with the stethoscope. Notwithstanding the obscurity of the diagnosis in this case, it was considered right to place this man under the influence of iodide of potassium, as a treatment likely to be useful, whether the

\* The preparation is in the Museum of the Royal College of Surgeons, Edinburgh.

tumour was solid, or was really an aneurism. Accordingly he got thirty-grain doses of the iodide of potassium twice a-day; he was placed on fish diet, a restricted amount of fluids, and confined to bed. The result was most encouraging; he got almost immediate relief from the agonising pain, while the uneasy pulsation felt by the man himself was also at once considerably lessened, and the force and fulness of the radial pulsations, as observed by us, seemed to be also greatly diminished. He continued steadily to improve; in a few months the tumour and bruit had completely disappeared, and on the 22d of January 1868 he was dismissed at his own request. He considered himself so well, that he engaged for a short voyage for the purpose of testing his reacquired health before finally proceeding to sea. This case is one, the obscurity of which is patent to all, and I am not disposed to press the diagnosis of aneurism; nevertheless, the symptoms pointed strongly in that direction, while the success of the treatment, and the manner in which the relief was obtained, seemed also to confirm it.

A year afterwards I reported the subsequent history of this case as follows. John Kerr, supposed aneurism of the abdominal aorta, left for a trial voyage, but never returned, and was given up as lost, and probably dead, when, in January 1869, the nurse got a letter from him dated Australia, and saying that he was now so well, nearly a year after his discharge, that he was about to give up the sea and go to the gold-diggings.\* Since then I have heard nothing of this patient.

The diagnosis in the next case was more unequivocal, and his history now also includes the *post-mortem* appearances, so that we know exactly what he did labour under, and to what extent it was remedied.

CASE XXXVIII.—James Wilson, aged 44, a mason from Newcastle, admitted into Ward VII. on August 31, 1867. About nine months before admission this patient began to have occasional attacks of lightness in the head, accompanied

\* *Edinburgh Medical Journal*, July 1869, p. 48.



with a flashing of light before his eyes. These attacks came on usually while he was at work, and obliged him to sit down for a little to recover himself. At first they occurred once or twice a-day, but they soon became more frequent, and he always felt much weaker after them. During these attacks he suffered from profuse perspiration. About the same time the patient began to suffer from "beatings" in his abdomen, in the left side of his chest, and on the right side of his neck. At the last-mentioned place a swelling appeared, which gave him great uneasiness, and produced a choking sensation. He consulted various medical men without relief, and at last came to Edinburgh, as already stated. On admission, it was found that he had no radial pulse in the left arm, but there was nothing to account for this, the "beatings" on the left side being apparently merely cardiac palpitation; on the other hand, those in the abdomen, and on the right side of the neck had each an abnormal and evident cause. About the lower part of the epigastric region, towards the left side, and lying close above the aorta, the course of which could be distinctly traced, a tumour, the size of a small orange, could be distinctly felt pulsating itself, and not merely moved by the artery beneath it; over this tumour a loud systolic bruit could be heard. On the right side of the neck there was also an evident pulsating tumour, extending up into the neck from the sterno-clavicular articulation, and towards the median line. This tumour was somewhat larger and longer than that in the abdomen, resembling in shape a large kidney potato. Upon any excitement, and especially when the man was up and walking about, its size increased considerably. No distinct bruit was heard over it, nevertheless it was evidently an aneurism implicating the innominate, subclavian, and carotid arteries. He was at once placed upon thirty-grain doses of the iodide of potassium twice a-day, which he continued to take steadily, with occasional intermissions, during his treatment, and a diet and regimen similar to that already described were prescribed for him; but as his symptoms were not

severe, strict recumbence was not insisted upon, and he was allowed to go to chapel every night. The intermissions in the use of the iodide were necessitated from the circumstance that it was not so well borne by him as by the other two patients; every now and then pain in the stomach, or severe headache, giving warning that it was time to stop it. These symptoms, however, always abated after leaving off the medicine for a day or two. His appetite was always good, but his bowels required to be regulated by medicine. No immediate effects were observed from the remedy, but after the lapse of some months, the abdominal aneurism was found to be quite firm and solid to the feel, while the bruit had disappeared, and could only be reproduced by pressing somewhat strongly with the stethoscope. After a time the aneurism in the neck ceased to swell out when he walked about; it also gradually became firmer, and he no longer suffered any inconvenience from it. The coats of the arteries in this position seemed to have undergone fusiform dilatation, and it appeared doubtful whether further treatment would be of any avail. His health was, however, much improved; he no longer had any disturbing pulsations; and though he occasionally suffered from lightness in the head, and dazzling flashes of light, his condition was unquestionably better than it had been, and considering his inveterate aneurismal diathesis, he probably reaped as much benefit from the treatment as was possible under the circumstances.

A year subsequently I reported of him as follows:—  
“James Wilson, labouring under an aneurism of the innominate, implicating the carotid and subclavian arteries, also an aneurism of the abdominal aorta, and a general aneurismal condition of the vessels. This man worked for many months comfortably, and without inconvenience, at his trade of mason, avoiding, as desired, any heavy lifts; he has now, however, got an easier berth in connection with the Caledonian Railway. His abdominal aneurism may still be felt as a hard, firm knot, now much diminished in size. His innominate



aneurism never troubles him, giving rise to no symptoms. But it is not consolidated; neither, however, is it any longer a true aneurism. At least it presents no symptoms of such a tumour, being to all intents and purposes apparently restored to the condition of an elastic artery, enlarged, but still an artery; fusiformly dilated no doubt, but no longer bulging as it did as a pulsating globular tumour stretching across the trachea, and no longer giving him any discomfort or uneasiness." \*

This poor man presented himself repeatedly for inspection and advice, and occasionally came into hospital for a week or two for relief of catarrhal symptoms, his aneurismal symptom remaining unchanged till the 27th of June 1870, when he re-entered hospital on account of a recurrence of the symptom of abdominal aneurism. The original aneurism was distinctly to be felt as a small hard nodule resting upon the aorta just beneath a large, soft, pulsating swelling, passing up beneath the ribs on the left side. Over this pulsating swelling a single systolic bruit was audible, the subjective symptoms were confined to flatulence and occasional pain of a dyspeptic character in the stomach. He was treated after the old fashion, with the iodide of potassium, and with a similar result; for by the beginning of December 1870 he was so well that I was thinking of discharging him, when he began to present symptoms of typhus,—a disease which, so far as ascertained, is believed to have been communicated to him by unauthorised and irregular contact with convalescent fever patients in the Infirmary chapel. For this disease he was removed to the fever wards, where he passed through the regular course of typhus, and after being disinfected, he was received back into a side-room seven weeks subsequent to his being transferred. On 24th January 1871 his abdominal tumour measured six inches, from the lower edge of the hard nodule of the former tumour to the point where it disappeared beneath the ribs. The tumour lay along the course of the aorta; it was three inches in super-

\* *Edinburgh Medical Journal*, July 1869, p. 48.



ficial breadth, and could be readily grasped through the thin parietes ; it was felt to be firm and devoid of lateral pulsation ; and unless very firmly pressed upon by the stethoscope, it conveyed to the ear no bruit whatever, merely a dull thud. At its upper extremity, however, an occasional and trifling systolic bruit was still perceptible, just where it dipped beneath the ribs, and this part seemed to have somewhat enlarged during his illness. A second time, therefore, this man was rescued from the jaws of death by this treatment. That it failed to wholly arrest the process need not, under the circumstances be wondered at, but we are rather disposed to marvel that so frail a body survived a disease to which so many powerful men have succumbed. Wilson made a good recovery, was discharged, and continued to maintain himself by manual labour till September 1873, when he was re-admitted to the Infirmary for a recurrence of his aneurismal symptoms, and he died there on the 14th of October. The large abdominal aneurism of recent occurrence—which occupied the whole of his left hypochondriac region—was at first fluid, and seemed to solidify ; at all events it became firmer under treatment, but it never underwent any diminution in size, though for this there was hardly time before he died exhausted. After death the whole of the aortic arterial system was found to be greatly atheromatous, the innominate, right subclavian, and right carotid arteries were thickened, and seemed somewhat larger than usual. The left subclavian was blocked by a firm fibrinous clot just where it passed out of the chest, the coats of the artery being contracted round this plug. The descending aorta gradually dilated, till, after passing through the diaphragm, it developed into an aneurism the size of a large cocoa-nut, beneath which could be felt the firm hard nodule of the original aneurism, which contained only *post-mortem* clots, but its walls were made almost as dense and hard as bone by atheromatous deposit.\*

\* This preparation is also in the Museum of the Royal College of Surgeons, Edinburgh.

CASE XXXIX.—Peter Reid, hotel porter, aged 46, admitted into Ward VII., June 18, 1868. This man was drunk when admitted, having avowedly taken spirits to nerve himself for his apparently rapidly approaching dissolution. He had intense dyspnoea, amounting to complete orthopnoea, violent harassing, but dry cough, and felt and looked as if on the point of suffocation, while a large, soft, projecting and pulsating tumour seemed to threaten him with death in another form. He was ordered at once fifteen minims of chlorodyne to be repeated every half-hour till the cough was quieted and also—

R Pot. iodidi,  $\mathfrak{zss}$ .

Infusi chirate,  $\mathfrak{zvj}$ . Solve.

Sig., One tablespoonful ( $\mathfrak{zss}$ ) three times a-day.

He was also, of course, desired to keep his bed, in which he was supported in a semi-erect posture, and to restrict his food and drink. Within twenty-four hours this man expressed himself as somewhat relieved, and in a few more he began to breathe more freely. He was an old soldier, and about twenty-four or twenty-five years ago had been cupped in a military hospital for palpitations, which continued more or less after his discharge, till about ten years past, in the end of February 1858, he came into this hospital on account of excruciating pain in the right side and down the right arm, which he had felt for some months previously. There was also at that time a slight swelling, accompanied by pulsation, to the right of the sternum. This pain was also most excruciating in winter when he caught cold or had any unusual exertion. After eighteen months the tumour protruded externally, and the pain then ceased to be so agonising. During the last eight or nine months the tumour has grown more rapidly than it has done for years, and this he attributes to the extreme violence of the cough. He has been under various physicians both here and in London, and has been, so far as he knows, mainly treated with digitalis and hyoscyamus internally, and the application of ice externally. To the right of the sternum

was a large pulsating tumour, extending from the third to the seventh ribs, and projecting fully an inch and a half beyond the level of the ribs. Part of this tumour was solid, but part, rather beneath the middle, was soft, painful to touch, projecting conically, and pulsating fluidly. Dulness extended for fully five inches all round the centre of the tumour, passing into the liver dulness beneath. The pulse in the right arm was smaller than that in the left. The heart's apex beat between the sixth and seventh ribs and one inch to the right of the nipple, but there was no apparent hypertrophy, and the cardiac dulness was about normal. There was displacement, but not enlargement, neither was there any abnormal bruit over either the heart or tumour. The right pupil was somewhat dilated. There was considerable pain in the side and down the right arm, but nothing to what it had been. After the patient had somewhat recovered from his frightful condition on entrance, a belladonna plaster was applied over the tumour, and the chlorodyne was gradually stopped as the cough ceased, which it had almost entirely done by the 12th of August, at which date the patient looked, felt, and expressed himself as much relieved; the pulsation in the tumour was much less forcible than formerly, and the tumour itself was flatter and more solid. On the 31st of August an ice-bag was substituted for the belladonna plaster, but was only continued for four days, because, although it felt comfortable to the patient, and relieved the distressing pulsations, it increased the cough very much; the belladonna plaster was, therefore, reapplied. At this date he expressed himself as feeling very comfortable, and able both to breathe and swallow easily, neither of which he could do formerly, the tumour also was gradually decreasing. On the 21st of September, however, the cough had again become very troublesome, accompanied by a distressing tickling sensation in the larynx, and an increase in the aneurismal pulsations, which were just as bad as they had formerly been. Chlorodyne made him sick, so he was ordered—



lessened by a retrogression to a whole bottle of pale ale on the 12th January, probably increased by a change to four ounces of brandy on the 25th of January, and only remedied by knocking off all stimulants on the 25th of February. I at once saw that no more good was to be done: the element of mutual confidence was lost. I therefore permitted Reid to rise every day and walk about the ward; the iodide mixture was continued, but the aconite was stopped; and I must say, that not one of my patients ever rose from their beds such a scarecrow as Reid did. This I attribute partly to the stimulants he had unfortunately obtained, and partly to the narcotics by which the evil results of his self-indulgence were so unavailingly sought to be neutralised. He was kept in hospital for three weeks longer, during which time he continued to improve, and was then dismissed on the 20th of March to go to his home in London. Thus this poor dying creature was, after nine months' treatment, dismissed in a comparatively active condition, and both looking and feeling well. A cast taken from the projecting tumour, about a month after his admission, and one taken just the morning of his discharge, show a very evident diminution in its bulk; while, in a letter which I received, 24th May 1869, from my former house physician, Mr Frank H. Hodges, he says—"I called on Peter Reid on the 28th of April, and found him in a very satisfactory condition; the tumour had considerably diminished in size (since his discharge), he was quite free from cough, and went out for a constitutional daily."

The next case is not so decidedly one of aneurism, but it is at all events one of aneurismal dilatation of the aorta, with diseased coats, and affords very evident proof of the relief obtained even in such cases from the treatment recommended.

CASE XL.—Thomas Moody, aged 39, a slater, from Cross-gates, Fife, admitted to Ward VII. on July 11, 1868. He stated that for sixteen months he had been complaining of severe pain across the upper part of the sternum, and a sensation of breathlessness. On examination, dulness was found to

extend across the whole of the upper part of the sternum. The cardiac dulness was normal, or very nearly so, apex beat between the fifth and sixth ribs. The first sound healthy, the second wanting, and replaced by a double bruit, loudest during the diastole, as heard at the aortic cartilage. This double bruit was propagated upwards along the arteries, but the systolic part of it was heard markedly louder and rougher over the left carotid artery than anywhere else. The finger, pressed deep into the trachial fossa, came in contact with a pulsating body. The other organs and systems were natural.

R. Pot. iodidi, ʒvj.  
Infusi. chiritæ, ʒvj. Solve.

Sig., ʒss. ter in die. To remain in bed, and to restrict himself somewhat in regard to his food and drink.

The patient very speedily expressed himself as greatly relieved; in a fortnight the rasping bruit over the left carotid was found to be much softened, the pulsation in the tracheal fossa was still perceptible, but not so much so as formerly, and he was discharged, at his own request, on the 3d of August 1868. There was in this case no history either of rheumatism or syphilis.

The next case, on the other hand, is not only a very well marked but also a very remarkable case of aneurism of the aorta, one which, even taken singly, would be sufficient to attract attention to the treatment propounded, but which, when regarded as only one of a series, provides indeed a very forcible illustration of the good effects to be derived from it.

CASE XLI.\*—Andrew Jamieson, a carter, aged 40, admitted into Ward II. under Professor Bennett's care, January 31, 1868. About eighteen months ago the patient had an attack of pleurisy in his left side; with this exception, he had been always healthy. About twelve months ago, without any known cause—as he is not aware of having strained himself

\* The history of this case is partly condensed from the Clinical Records of Ward II.

in any way, though constantly in the habit of lifting heavy weights into his cart—he first felt a severe and constant aching in the left side of his chest, over a spot about the size of a crown piece, situate about two inches above the nipple. This pain was much increased on making any exertion or on stooping. To relieve it he applied several mustard poultices, and took cod-liver oil, without, however, deriving any benefit. Notwithstanding the pain he suffered, he continued to work up to July 1867, but he was then compelled to give it up entirely on account of his sufferings. The pain was now more severe than ever; his breathing short and wheezing; he had a troublesome cough, worse on making any exertion; he experienced a choking feeling when he stooped, and he felt a difficulty in swallowing any solid food, which seemed to stick opposite the upper margin of the sternum. His voice also now became weak and somewhat hoarse—*vox anserina*—and he felt a disagreeable pulsation in his chest. For these symptoms he unavailingly sought relief in the Glasgow Infirmary, and finally came to Edinburgh. Up to the period of admission the pain had spread very much over the upper part of the chest, but he does not think that the other symptoms had increased; he has never had any headache or hæmoptysis. The pulse at the left wrist is almost imperceptible. The right pulse is 84, of moderate strength, and slightly jerking. Cardiac dulness, not noted. The heart's apex beats three inches below, and a little external to the nipple line. Only the first sound is heard at the apex; both sounds are heard at the base normal. Over the upper part of the left side of the chest, anteriorly, there is a distinct bulging, most marked over the second rib and second intercostal space. Over a spot about the size of a crown piece there is a distinct impulse communicated to the stethoscope; over the left side there is dulness on percussion from the clavicle to within two inches of the nipple line, extending across to the opposite sternoclavicular articulation (*vide* fig. 26). Over this dull area a double blowing murmur is to be heard, “the second sound,



however, being very feeble, and heard most distinctly over the sternum, opposite the articulation of the second rib, also heard over the great vessels at the root of the neck."\* At the right apex the inspiration is harsh and the expiration prolonged; over the left apex, anteriorly, the double murmur referred to completely obscures the sounds of respiration, and are everywhere else normal. The patient has a very loud and frequent cough, of a clanging metallic character, without expectoration. He complains of severe dull aching pain on the left side of the chest, from below the clavicle to the level of the nipple; this occasionally extends to the right side. He gets only occasional short snatches of sleep, owing partly to the pain referred to, and partly to the frequent recurrence of a choking sensation referred to the throat. His tongue is clean, appetite bad, but bowels regular. Urine, sp. gr. 1·030, with deposit of urates, but otherwise normal. There is a slight puffiness of the integuments over the upper part of the sternum, and the veins over the dull area are visibly enlarged. He was ordered steak diet (Royal Infirmary diet tables), and—

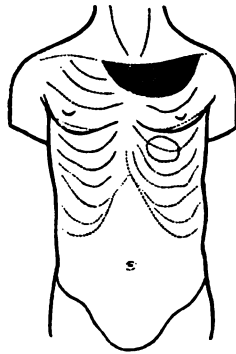


Fig. 26.

R Tinct. ferri perchlor., ℥j.  
Aqua, ℥vj. M.

Sig., One tablespoonful three times a-day.

February 4th.—The patient continues much the same as on admission. He still chokes on his food, and requires a "drink to wash it down." His cough and spasms of choking are very violent, and of frequent recurrence. Ordered to take fifteen minims of sulphuric ether in water, when the cough comes on.

12th of February.—Pain and cough both worse; ordered—

\* I here give the *ipsissima verba* of the report. The second sound refers, I believe, to the diastolic blowing heard over the tumour, but the expression is ambiguous.

R Tinct. camph. co., ℥j.  
Mucilaginis, ℥j. M.

Sig., ℥j. pro dose, *pro re nata*.

He continued not certainly improving till the 17th of February, when it is stated that the pain is better than it has been; slept two or three hours in the night. Complaints of difficulty of breathing, much worse if he makes the slightest exertion. Pulse 100, of moderate strength; ordered—

R Potassii iodidi, ℥ij.  
Aquæ, ℥vj. Solve.

Sig., Two tablespoonfuls three times a-day.

Next day, 18th of February, the pain is said to be not so bad as it had been; the pulse was 88, and of moderate strength; the cough and dyspnoea still continued. He was ordered to take fifteen minims of chlorodyne in water at bedtime. On the 22d of February it is reported that he did not cough so much last night, and that the pain no longer passes across the chest, but is confined to one spot, the size of a crown piece, between the clavicle and nipple; the pulse is 84, and of moderate strength. On the 3d of March, it is reported that the cough was more troublesome during the previous night, and that he complained of slight soreness across the lower part of the chest on coughing. Ordered to discontinue the iodide of potassium mixture. On the 5th of March the patient was ordered full diet instead of steak diet. On the 10th of March, his cough becoming very troublesome, he was ordered—

R Sp. chloroformi, } ℥iv.  
Chlorodynii, }  
Aquæ, ℥vss. M.

Sig., One tablespoonful when the cough is troublesome.

The patient said the pain was worse in the left side of the chest. He thought it had not been so well since he left off the iodide of potassium. Ordered to recommence his mixture, but to take only one tablespoonful three times a-day, so that he then got only ten grains of the iodide for a dose. He

continued much the same, not complaining of the pain till the 21st, when he was ordered to discontinue the iodide of potassium mixture. On the 23d of March, it is reported that the patient says he cannot see distinctly when reading or writing, he feels as if a mist came before his eyes; he has noticed this since the 19th. On the 31st of March, he was ordered to apply pounded ice, in a bag of thin gutta-percha, over the pulsating tumour, on the left side of the chest, for one quarter of each hour during the day. On the 16th of April the cough was rather worse, and he was ordered to resume the iodide of potassium, in twenty-grain doses three times a-day. At this date the dimness of sight formerly complained of is reported better. On the 24th of April the iodide again was discontinued, because of slight watering and weakness of the eyes. On the 28th of April, it is reported that the cough recurred with great violence during the preceding night, with great difficulty of breathing, loud snoring noise heard over all the chest posteriorly, with a rattling in his throat; no dulness posteriorly, vocal resonance increased equally on both sides. Ice to be discontinued. Ordered to recommence the iodide of potassium in twenty-grain doses three times a-day. On the 29th of April he was ordered—

R Spt. æth. sulphurici, ℥ij.

Chlorodyni, ℥ij.

Decoct. senegæ, ℥vj. M.

Sig., One tablespoonful when the cough is troublesome.

April 30th.—The breathing is said to be not so difficult, his cough continues, the expectoration frothy and purulent. On 8th May, it is stated that the area of dulness remains unchanged; the sounds heard over it are, however, much diminished in intensity, and the blowing is scarcely audible. Percussion over the lungs is normal, but loud sonorous râles are everywhere audible, with prolonged expiration, especially on the right side posteriorly. The cough is not so harsh and barking as formerly, but it is now accompanied by a most



copious purulent expectoration, especially at night. All his medicines to be stopped except the iodide mixture, and to have the following draught :—

R Chlorodynii, ℥x.  
Spt. æth. chlorici, ℥ss.  
Mucilaginis, ℥j.  
Aquæ, ℥x. M.  
Ft. haustus nocte sumendus.

On the 22d of May he was ordered a hot poultice, on account of a soreness in the chest, most felt after coughing.

9th June.—Since last report he has continued much the same. He sometimes complains of pain in the chest, still has cough, with profuse purulent expectoration, and loud sonorous râles, both anteriorly and posteriorly. He still continues to take the sedative draught and the iodide mixture. Upon percussion, the dulness is found to have extended more towards the right side, now reaching to about the middle of the clavicle (*vide* fig. 27).\*



Fig. 27.

On 23d June, it is stated that the patient complained of great pain in the chest, which seemed to be increasing, and for which he had poultices frequently applied. He also complained of great weakness, for which he got two ounces of port wine daily. His breathing was however easier, the cough and expectoration greatly diminished. The pulsations over the

now greatly enlarged dull area are however increased, and the double blowing murmur is more distinct than formerly. Posteriorly, the blowing murmur can also be distinctly heard about the sixth dorsal vertebra. 20th June.—Complains of pain in the left side being increased. Poultices ordered. 27th June.—Still complains of great pain in his left side.

\* Figures 26 and 27 have been accurately copied from drawings made at the time by Dr Bennett's clerk. The dark spot points to the place where the red projection subsequently to be mentioned was thrown out.

Twenty minims of a solution, containing nine grains of hydrochlorate of morphia to the ounce, were injected subcutaneously over the painful part. He continued to improve slightly. On 16th July, it is stated that the patient feels very well, has little cough or expectoration; he takes his food well, and has very little pain. On 23d July, it is stated that the patient continues better, his breathing easier, cough and expectoration almost gone, and for the last two or three days he has been getting up and walking about the ward, though unable for exertion, or even for much gentle exercise. He continues the iodide of potassium. At this time, Ward II. being shut for the autumn, he was transferred to my care in Ward VII. Shortly after being sent up to me he walked through the whole ward to the water-closet, which is somewhat draughty, and back again, and was thereupon seized with a return of his cough, copious purulent expectoration, and violent pain in the left shoulder, and over the large pulsating tumour, on the left of which a small projection about an inch square was thrown out, over which there was a red blush. He was strictly confined to bed, a belladonna plaster applied over the tumour, with half a drachm of the iodide of potassium in infusion of chiretta, three times a-day, also—

R. Syrupi scillæ, ℥ij.  
 Spt. lav. co.  
 Tinct. opii ammoniatæ, } aa ℥iv.  
 Syrupi simplicis, ℥j.  
 Aq. menth. piperitæ, ℥ij. M.

Sig., One tablespoonful every three hours, and fifteen minims of chlorodyne to be given additionally when required.

In a few days he was relieved. The projection subsided and he seemed going on favourably, but the annual cleaning of the wards then came on, and on the morning of the 13th of August he was unfortunately permitted to walk down two stairs, each one storey high, to Ward No. 1. This, however, he seemed to bear pretty well; he expressed himself as none the worse, but his cough was most certainly not improved.

Upon his return upstairs, a week subsequently, I had him carried in a chair, but the exertion, and possibly the somewhat damp air of the newly-washed ward, were too much for him; a violent exacerbation of his cough took place, accompanied by a great increase of the size of the tumour, which was covered by a red blush; there was complete loss of the pulse in the left arm, accompanied by coldness and excruciating pain in the limb; his expectoration was copious and purulent, and occasionally both streaked and stained with blood. His left arm was ordered to be swathed in flannel, and he to continue his cough mixture, to have also fifteen minims of chlorodyne *pro re nata*, to have his iodide mixture four times a-day, and to have a large belladonna plaster applied over the whole tumour. For many days he took two drachms of chlorodyne daily, besides his cough mixture—indeed, the orders were to give him a full dose of chlorodyne at once when required, so as at all hazards to keep the cough quiet. He bore this treatment well, his tongue kept clean, and his appetite fair; in eight days the expectoration began to lessen, and the cough and pain to cease. By the 31st of August they were almost gone, and the expectoration reduced to a trifling amount of mere mucus; the arm was again warm, and feeling like itself; the pulse restored to the wrist, though still very feeble; the tumour somewhat diminished in size, and apparently not pulsating so strongly: he was lying quietly in bed reading, and expressed himself as feeling quite a new man. After this he was kept constantly lying on his back in bed for the next nine months; for the greater part of this he was on a water-bed; he had occasional relapses of his cough, especially during the changeable weather of autumn and early winter, for which a tolerably free use of chlorodyne or other opiate was required; but there were no more threatening symptoms in regard to the aneurism, which continued steadily but slowly to improve. During all the next nine months, he continued to take two drachms of the iodide of potassium daily, with the exception of twice, when



it was omitted for a day or two on account of gastric irritation, as evinced by pain and vomiting. At last, towards the end of May, the tumour seemed sufficiently consolidated, and all the symptoms so moderated, as to warrant the patient's being allowed to get out of bed. Although he looked well and healthy, yet I considered it proper to prepare him for his exertion, by adding one drachm of the iodide of iron to his iodide mixture; this he took from the 18th to the 21st of May, when he was allowed to get up; the iodide of iron was continued in combination with infusion of calumba, and the iodide of potash omitted. I find I have omitted to state, that when I first made up my mind to allow Jamieson to rise, finding his pulse rather more rapid than I liked, which I now believe was partly the result of a fearful joy, I put him for several days upon gradually increasing doses of tincture of aconite, along with his iodide mixture; this having been pushed as far as I thought proper, without any decided lowering effect, I then placed him, as I have just stated, upon the iodide of iron. At this time his pulse was 100, much fuller in the right arm than in the left; the skin moist; his cough so slight as to be inappreciable, never heard during visit; his expectoration merely a trifling catarrhal mucus; his voice still thin, and somewhat feeble but distinct, and very different from what it was; respiration everywhere natural, and the percussion sound posteriorly normal. Anteriorly, the apex beat was feebly felt between the sixth and seventh ribs, a little outside the nipple line, the heart being probably elongated, and certainly displaced, the lung covering it. Just outside of the left edge of the sternum, dulness extends from the liver quite up to the lower border of the first rib. In the nipple line, dulness extends transversely to a distance of two inches from the left edge of the sternum. Just at the inner edge of the left clavicle there is a small patch of clear sound; with this exception, dulness extends along both clavicles, from about one inch and a half to the right of the right edge of the sternum, to about four inches to the left of its left edge. The dulness

on the right side is bounded by a semicircular line joining the sternum at the upper border of the second rib. On the left side the dulness is also bounded by a semicircular line, the centre of which is about the middle of the clavicle, and in this position it does not descend beneath the middle of the third rib. The sternal ends of both clavicles are dislocated, the right to the extent of half an inch, the left to about a quarter of an inch; on the upper part of the sternum there is a considerable puffiness, and over it, and over the dull portion to its left the veins are enlarged and tortuous. A finger placed in the tracheal fossa feels a solid mass behind the sternum. Over the apex beat, the first sound of the heart is audible, somewhat obscured by the rough systolic portion of a double bruit, heard loudest at the middle of the sternum, just at the junction of the fourth rib; this is propagated upwards loudly and roughly into both carotids; less so into the right subclavian, while over the left subclavian, in the dull portion already mapped out, we have a solid tumour, pulsating, but not very forcibly, and only with a movement of elevation, and none of dilatation, over which no bruit whatever is heard, merely a dull thud. The patient has now no difficulty in breathing or swallowing: he walks up and down stairs, and about the airing ground; he says he has now no cough. The aneurism seems still going on consolidating. I have again placed him for precaution's sake upon the iodide of potassium, but he is now ready, and I believe fit, to be discharged. I shall, however, protest against his testing his power of procuring a livelihood for himself by a return to a laborious occupation. I sent this patient shortly after this downstairs to Dr Bennett, who exhibited him to his class, and, I am told, expressed himself satisfied with the reality of the improvement which had taken place.

Jamieson was shortly after discharged. He went to the workhouse, where he remained till the 1st of November 1869, when he was re-admitted into Ward V. for some catarrhal symptoms. His aneurism remained in *statu quo*. A thick

œdematous collar full of tortuous veins had developed at the lower part of the neck and over the upper part of the sternum. He was discharged in April 1870, and went to Glasgow, where he remained much the same when last heard of.

This very interesting case affords an instructive example of how much may be done by appropriate treatment even in the most unfavourable cases of aneurism. And it speaks well for the treatment under consideration, that it was capable of relieving symptoms so severe as those of Jamieson, and of prolonging life in circumstances so apparently hopeless.

CASE XLII.—William Allison, aged 22, a tailor, from Glasgow, a native of Dumfries, was admitted into Ward VII. on August 3, 1868, labouring under severe cough, with copious purulent expectoration, having also a pulsating tumour in the left side of the chest. He was a thin, worn-looking man, and stated that he had been nine months ill, having already sought relief in the Glasgow Infirmary for a severe cough and pains in the left side, especially in its lower part. There he was under Dr Steven's care, who discovered the aneurism, the existence of which was unknown to the patient. No special treatment was used for the aneurism; his strength was supported, and cough soothed. He remained in the Glasgow Infirmary from the 7th of March till the 20th of June, when he was discharged, improved as regards his cough, to the Convalescent Hospital at Bothwell. From thence he went to Dumfries, where he became worse, was advised to come to Edinburgh, and was sent to me by Dr Joseph Bell, whom he had consulted. On admission, he was found to be exhausted by a constant harassing cough and copious purulent expectoration. He had also suffered much from severe pains in the left side of the chest, extending over the shoulder and down the left arm to the elbow, also up the left side of the neck to a limited extent. Pulse 110, rapid and feeble. Apex beat between fifth and sixth ribs, and slightly to the left of the nipple. The cardiac dulness did not, however, seem increased. The heart seemed rather pushed to the left, and



somewhat backwards. The heart's sounds were normal, but fainter than natural over the apex; the second somewhat accentuated at the base. Between the second and third ribs on the left side there was a conical elevation, rising about half an inch above the level of the ribs, pulsating fluidly, with thin walls and a distensile action. This pulsating tumour measured transversely about an inch and a half, while another inch and a half of dulness extended to the edge of the sternum; it passed upward to the lower border of the first rib, and below it was lost in the cardiac dulness. Over this region the pulsatile wave passed from right to left, and a tolerably loud and well-marked bruit was to be heard, terminated by the accentuated thud of the closure of the aortic valves. This was most distinct close to the sternum; more to the left this thud appeared to be replaced by a second blowing sound. The right lung was normal both as to percussion and auscultation; the left lung was dull anteriorly, and over its lower half posteriorly, the respiration being obscured apparently by thickening of the pleura (the remains of an old pleurisy which he had in Glasgow), sonorous and creaking râles being heard there, as also over the apex. Right pulse perhaps a shade fuller than the left. Pupils both natural. To have a belladonna plaster applied over the tumour, and—

- R. Morphæ hydrochloratis, gr. j.  
 Acidi hydrochlorici dil., ℥ v.  
 Acidi hydrocyanici dil., ℥ss.  
 Syrupi scillæ,                    }   ℥℥ ʒj.       M.  
 Aquæ fontanæ,                }  
 Sig., One teaspoonful occasionally.
- R. Potassii iodidi, ʒvj.  
 Infusi chirate, ʒvj. Solve.  
 Sig., One tablespoonful three times a-day.

The pain ceased in a few days. By 1st September the cough was almost gone, the sputa nummular but scanty, his breathing easy, and he felt altogether more comfortable. His pulsating tumour scarcely projected at all, and felt somewhat

firmer in its walls; his pulse 96. By 30th September the cough and expectoration had both entirely ceased, and on 30th October he left, thinking himself quite cured. The pulsating tumour was lessened, but its walls were not thickened, and the bruit was as loud as ever. About a month afterwards I had a note from him requesting re-admission, which I granted, upon condition that he should agree to confine himself to bed for six months at least, and upon 10th November he was re-admitted at his own urgent request. His cough was now very harassing, and he expectorated about 5xv. of purulent matter during the night alone. He was placed on his former remedies, but on the 23d of November he said that he vomited the iodide. He was ordered to continue the iodide mixture as formerly, but to have ten minims of chlorodyne *pro re nata*, instead of his cough mixture. 24th November.—It is reported, "no more sickness." On 28th November he was so exhausted with his purulent expectoration that I was forced to give him four ounces of brandy in the day. At this date his right lung was found to be normal. On the left side anteriorly there is complete dulness everywhere, except at two points—*first*, just below the left clavicle, and for about an inch from the sternum; there the dulness is not quite complete: *second*, there is a patch of resonant lung, bounded above by the clavicle; anteriorly, by a perpendicular line from the centre of the clavicle to the upper border of the third rib; posteriorly, by the anterior fold of the axilla, down to where this joins the fifth rib; and inferiorly, by a semicircular line joining the two inferior points. The apex beat is between the fifth and sixth ribs, two inches to the left of the nipple, and one inch and a half below it. The heart sounds distant but normal, at least free from bruit, at the apex. The base is so covered up by a large pulsating tumour that the sounds proper to it cannot be distinguished. This tumour pulsates visibly and fluidly between the second and third ribs, commencing immediately within the line bounding the resonant space just mentioned, and extending to the sternum; the pulsatile and distensile wave passes from



right to left, and seems to follow the heart's impulse very closely; the interval being distinct, though brief. Over the whole dull portion of the chest anteriorly—chiefly, however, heard where the pulsatile tumour exists, and specially well marked close to the left edge of the sternum—there is a loud bruit, seeming to follow the loud accentuated second sound, and there is also a thrill most evident at the close of the pulsatory wave. Laterally the percussion sound is somewhat dull. Posteriorly there is no dulness above the centre of the scapula; beneath that the dulness is well marked. Anteriorly, over the sonorous patches, there are moist rattles and creaking sounds. Posteriorly, as low down as the middle of the scapula, the respiration is normal, with a few rattles; no vocal resonance. Beneath that the vesicular respiration is faint, almost inaudible, and masked on deep inspiration by coarse crepitation. From the fourth dorsal vertebra, as low down as the seventh, and for four inches to the left of the spine, the pulsation is distinctly audible, but no bruit.

By 24th December his cough and expectoration were quite gone. He was looking well, gaining flesh, and the pulsations were much quieter. By the 26th of March he was so much recovered, and the pulsation so quiet, the walls feeling so solid and dense, that I ventured to express a hope that I should soon be able to let him out of bed. Unfortunately, that very afternoon he rose without leave, and left the hospital. Of course, upon his return, he was very properly dismissed for his misconduct by the house physician, and I heard nothing of him till August 1869, when Allison was re-admitted into Ward V., his aortic symptoms being similar to those already described, while his lung symptoms were much aggravated, and there he remained, getting gradually weaker, till his death on the 27th May 1870. He was always an unmanageable patient, and reaped but little benefit from treatment. He died from exhaustion (asthenia) consequent on the copious suppuration of the left lung. At the autopsy, on the 30th May, the aorta was found much dilated and atheromatous, an aneurism the size



of a cocoa-nut was found to extend from just above the aortic valves to the commencement of the descending portion of the arch, occupying the whole circumference of the aorta. The aneurism consisted of two sacs, the one bulging anteriorly in the concavity of the arch, the other posteriorly. The two sacs were separated internally by a groove corresponding to the convexity of the arch. The coats of the sacs were calcareous, the interval between them comparatively healthy. A firm decolorised clot, two and a half inches long by two and three-quarters broad, occupied the anterior sac. The descending aorta curved abruptly from the aneurism by a circular opening the size of the normal aorta. The right lung was healthy; the left lung greatly atrophied, condensed, collapsed, and filled with suppurating cavities. The liver weighed seven pounds fifteen ounces, and was firm and waxy. The spleen weighed fourteen ounces, and was waxy, the Malpighian bodies notably so. The kidneys were also waxy, but not remarkably so; they were also somewhat fatty. The intestines were glued together by lymph recently effused; the colon was greatly distended. The two sacs of this aneurism, of which the left was the largest, communicated so freely, that it was only anatomically, and by reason of being separated by the healthy patch of aorta, that they could be called two. There was a large bulging to the left, and a smaller one to the right, or rather more anteriorly. It seems impossible to account for the diastolic character of the bruit, which was distinctly recognised by Dr Steven of Glasgow, under whose care he was before coming to Edinburgh, unless we suppose the sac to have still retained some contractile force, and to have thus produced it by its own systole. Some support is given to this view by the fact that the thrill was most marked at the subsidence of the systolic wave. As to the singular delayed character of the impulse which followed that of the heart by a distinct though brief interval, there can be no doubt that this phenomenon, so rare in aneurism, was caused by the large size of the sac and the very rough character of its interior—rough-

fectly free from pain and every aneurismal discomfort, and expressed his determination not to go to work for a few days. Dr M'Bain wrote me in May 1867, that he was working as a scavenger without inconvenience; that there was still excessive pulsation, but that the tumour had almost disappeared. On the 15th of February 1871 this man was exhibited to the Edinburgh Medico-Chirurgical Society; the tumour, still firm and solid, was then preceptibly diminished in size, scarcely projecting above the level of the sternum, while the man was strong and healthy, and totally free from every discomfort.\* He is still (1875) alive, perfectly well, and quite able for his work.

I may here mention that I have successfully treated in a similar manner many other cases of innominate aneurism, which it would be superfluous to relate more particularly. In three of these cases the cure seemed to be considerably expedited by the external application of iodine liniment over the tumour, which was ultimately reduced to the dimensions of a somewhat dilated but firm and no longer distensile artery. The aorta was implicated in one at least of these aneurisms, and in another it was so much so as to constitute the more serious part of the disease; nevertheless, the last time I saw him the tumour was firm, and the distensile pulsations had ceased. In the diagnosis of innominate aneurisms, it is right to remember that even in the normal condition the artery sometimes divides considerably above the clavicle,† and then when only moderately dilated it simulates an aneurism very efficiently. In one case, in which after death the artery measured only one inch and three-quarters, and did not appear to be abnormally dilated, the appearance of an aneurismal tumour was so efficiently simulated, that Mr Annandale was consulted as to the propriety of operative interference.‡ Such a case would probably have been rapidly cured by the

\* *Edinburgh Medical Journal*, April 1871, p. 935.

† Quain's *Anatomy of the Arteries*, London, 1844, plate xx. fig. 3.

‡ *Edinburgh Medical Journal* February 1871, p. 739.

external application of iodine. In the cases I have mentioned, the abnormal bruits, and the implication of the aorta in some of them, left little reason to doubt the actual existence of aneurism; at all events, in diagnosing the cases these various sources of fallacy were carefully considered, and rejected as insufficient to explain the phenomena present.

CASE XLV.—Samuel Moore, aged 47, an old soldier, now a stevedore. He was wounded in the Crimea, and was subsequently for nine years in India, where he enjoyed the best of health, having during all that time only once had a week's illness. His habits are, however, confessedly intemperate. He was admitted into Ward VII. on the 14th September 1868, and stated that he had caught cold from getting wet when working very hard at the quay of Glasgow, about seven weeks previously. A violent cough then came on which had lasted four weeks, when being seized with a violent fit of coughing while wheeling a barrowload of cheese from the ship's side to the quay, he felt a sudden pain shoot through his chest and down his left arm, which immediately began to swell. On admission, his right pupil was found to be slightly dilated, and the right radial pulse a shade weaker than the left. The left arm was swollen, measuring round the middle of the biceps twelve inches and a half, the right one only measuring eleven inches. The upper part of the left forearm measured also twelve and a half, the right one only ten inches and a quarter. The veins on the anterior part of the left chest, shoulder, and arm, were tortuous and much enlarged. The apex beat was between the fifth and sixth ribs, directly beneath the nipple. The heart's impulse and dulness were normal, the dulness however, extending to the upper part of the sternum, and entirely across it. The systolic sound in the mitral area was rather shorter, and apparently not so full as usual. The diastolic sound in the aortic area was somewhat accentuated. A rough systolic bruit, closed by the accentuated diastolic sound, is to be heard in both carotids, but loudest and roughest in the right one. Pulsation is to be felt in the supra-



sternal notch. Percussion over the lungs was natural, but loud sonorous râles were to be heard all over them. He had a violent cough and copious frothy expectoration. He had great pain shooting down the left arm, round the left shoulder, and up into the neck. Ordered a squill and opium mixture and half a drachm of iodide of potassium, in infusion of chiretta, three times a-day. In this case the diagnosis of aneurism was at first somewhat obscure; the patient, however, improved under the treatment; the swelling of the arm, the enlargement of the veins, the cough, and the expectoration all gradually lessening, and the rough bruit in the right carotid becoming apparently softer. I had, however, ceased to look for any more definite signs of an aneurism, when one day in the beginning of November, happening to place my hand upon his chest, I felt a well-marked pulsating tumour just to the right of the upper part of the sternum. Being hurried at the time, no careful examination was made for a couple of days, but by that time the very evident and well-marked pulsating tumour, which was readily felt by myself and others, had ceased to be so easily perceived. There was, however, a dull patch between the first and second ribs, extending for about an inch and a half to the right of the sternum, and reaching from the upper edge of the second to the middle of the first rib. Over this dull patch pulsation was only very obscurely to be felt. The accentuation of the aortic sound was very well marked over the dull patch just referred to, preceded by a loud whiz, extending up into the right carotid and somewhat into the subclavian, but not well marked there. The aneurism seemed to be somewhat movable; because when pulsation was so evident to the right of the sternum, it was not so perceptible in the tracheal fossa, and *vice versa*. The swelling of the arm had quite disappeared, and all his symptoms were so much alleviated that he considered himself quite well. Unfortunately, he had to be dismissed for misconduct on the 28th of November. He subsequently led a life of alternate dissipation and medical treatment, not under my care, however; and on the

12th of June 1869, he presented himself to me and besought me for re-admission. He then had a large pulsating tumour on the right side, about which there could be no mistake. I neglected to mention that this patient had tolerably smart symptoms of iodism, pain in the head and coryza, when he first commenced to take the iodide, but a steady persistence in the half-drachm doses speedily established tolerance.

On his first admission the symptoms in this patient presented some obscurity, and many of those who saw him then were disposed to regard the swelling of the arm as due to the presence of enlarged glands, because of the apparent absence of all the more definite symptoms of aneurism. The very sudden manner, however, in which the symptoms occurred was conclusive to my mind against the theory of any solid tumour, and this, coupled with the rough bruit loudest in the right carotid, led me to hold to the theory of aortic aneurism as being a more probable explanation—an explanation which the sequel proved correct. It is somewhat remarkable that while the bruit was loudest in the right carotid, the venous swelling was confined to the left arm. It seems probable that the aneurism arose from the upper part of the ascending aorta, just before the giving off of the innominate; that it was distinctly saccular, seated on the artery as it were by means of a neck, and therefore mobile; and that it only pressed slightly upon the left innominate vein, so that the swelling of the arm was due to position, coupled with the effect of an obstruction so slight as not appreciably to interfere with the venous circulation of the head and neck, the return of the blood from which was favoured by position as much as that from the arm was hindered. The position of the aneurism when it did manifest itself, and its subsequent disappearance, are strictly consistent with this view. I may add that not long since I had occasion to see an aneurism, not known to be the result of accident, in which the primary symptoms were precisely similar, and the seat of the tumour when it did become manifest was exactly the same. This case

had been treated on Valsalva's principles, with great temporary relief, but all the symptoms returned with returning strength. The iodide of potassium gave speedy relief to the pain, but I am not aware of the ultimate result of the case.

Moore acted up to his character, and kept loafing about the hospital for a time. Latterly he has quite disappeared; most probably his disease has proved fatal.

CASE XLVI.—William M'Alpine, a hawker, aged 41, admitted into Ward VII. on June 1, 1868. This is the case referred to by Dr Warburton Begbie, at p. 1071, *Edinburgh Medical Journal* for June 1863. For about six years, therefore, he had been labouring under symptoms of aneurism, while during that time his symptoms were relieved, and kept more or less in abeyance by the irregular use of the iodide of potassium, taken chiefly at his own hand. On admission, the percussion on the right side was found to be normal. On the left side, a dull patch extended from near the right side of the sternum to a distance of four inches along the upper border of the third rib, close to the left side of the sternum. This dulness extended upwards to the lower border of the first rib, while beneath it was lost in the cardiac dulness; towards the left it rounded off semicircularly. The ribs over this patch seemed to protrude. The cardiac dulness was normal, but no apex beat was to be felt. The cardiac sounds over the normal position of the apex were heard more distant than usual, the second sound was heard distinctly accentuated in its normal position. Over the dull area already described, the heart sounds were heard louder, and more accentuated than normally; no bruit was to be heard, but a distinct pulsatile movement was communicated to the ear, though it was not so perceptible by the hand. The right radial pulse was fuller and stronger than the left. Pupils both alike. Posteriorly, on the left side, a distinct pulsatile thrill was communicated to the ear, but none was to be felt by the hand, nor did percussion bring out any appreciable dulness. The annoying sensation of pulsation was,



however, only too perceptible to the patient himself, who said it prevented him leaning back upon a chair. A fine fringe of vascularity ran along the lower border of the thorax from one side to the other, and there was considerable œdema of the lower extremities and abdomen, which had existed for some time, and which he had been in the habit of relieving by purgatives. No appreciable ascites. Urine normal. To have half a drachm of the iodide of potash in infusion of chiretta, three times a-day. To be strictly confined to the recumbent posture, and ordered to restrict himself both in food and drink. On the 29th of August, he expressed himself as much relieved, the distressing sensation of pulsation being no longer perceived by him. The œdema, however, troubled him much, and for this he had several purgatives, which ultimately relieved him. He was dismissed on 16th September, at his own request. He again returned on the 26th September, for a cough, and remained in hospital till 23d October, taking the iodide mixture as formerly. He was then dismissed at his own request; the pulsation was no longer perceptible to himself, and only very faintly appreciable on auscultation; the pulsation anteriorly over the tumour was also much less perceptible. Again he returned about the end of February 1869, not now complaining of his aneurism, which remained in *statu quo*, but of intense œdema of the lower limbs and body, with some ascites, and also oppression of breathing. The upper limbs and face were comparatively free from swelling. There was no albuminuria, but purgatives and diuretics alike failed to give relief, and he was evidently sinking from the dropsy, and apparently had not many days to live, when he died suddenly, suffocated by hæmorrhage, the blood escaping from his mouth.

The autopsy took place on the 7th of March. The cutaneous tissues were œdematous and extremely congested. The face was livid. Upon opening the thorax the sac of the aneurism was found firmly adherent to the left margin of the sternum, and to the third left costal cartilage. Opposite this point externally there was a slight elevation. The right pleura contained a

large amount of reddish serum. Between the two layers there were several patches of dense old adhesions. The lungs were compressed, particularly the lower lobes. The left lung was firmly adherent to the walls of the thorax and to the surface of the aneurism. Part of the lower lobe of the right lung was also connected to the surface of the aneurism. The pericardium was greatly thickened. The heart was small; its cavities not dilated; the valves were competent. There was no marked pressure on the trachea, bronchi, or œsophagus. The branches of the arch of the aorta were atheromatous, but not compressed. Neither the pulmonary arteries nor any of the large veins were compressed. The coronary arteries were freely open, the aortic valves competent. The whole of the ascending part of the arch of the aorta had its inner coat atheromatous and calcareous. The middle coat was greatly thinned. The mouth of the aneurism was in the anterior wall of the aorta, about one inch above the valves. It measured three inches vertically, and nearly two inches across. The aneurism projected from its point of origin forward and toward the left side. Transfixed at its greatest breadth it measured four inches and a half, and from above downward five inches. Its anterior part contained firmly adherent fawn-coloured clots; its posterior part contained a large, softer, and more deeply coloured clot. On the same level as the aneurism just described there sprang another from the right side of the aorta, with an orifice about the size of a shilling, the whole tumour being about the size of a walnut. This second aneurism pressed upon the right auricle, the muscular fibres of which were much hypertrophied. The chief veins were free. There was slight flattening of the left vagus nerve on the surface of the aneurism. The liver was slightly cirrhotic, its surface studded with numerous miliary granules. The kidneys were natural. This small aneurism which, doubtless, by its pressure on the right auricle, was the cause of the dropsy, and which, being continually churned by the greatly hypertrophied right auricle, could never be kept sufficiently at rest



to induce the formation of a clot in it, was ultimately the cause of death, rupturing into the lower lobe of the right lung. The parts were exhibited at the meeting of the Medico-Chirurgical Society on the 7th April, as an apt illustration not only of one mode of cure of an aneurism, but also of one of those conditions—and that not a common one—which too often render our best-devised plans of cure abortive.

The next case which I shall give is one which illustrates the effect of this mode of treatment in a rarer form of aneurism—weeping aneurism.

CASE XLVII.—Thomas Simpson, a cabman, aged 58, was admitted to Ward IX. of the Royal Infirmary on June 26, 1869, on the recommendation of Dr Joseph Bell.

The patient states that up to his thirty-fifth year he had been perfectly healthy. At that time, while attending a yeomanry drill, a carbine was fired close to him, which caused the horse he was riding to rear. The animal fell on the top of him, and crushed him. On being extricated, he was faint and unable to stand. On medical assistance being procured no injury could be detected beyond a very severe bruise between the shoulder blades. This accident confined him to bed for three months, and for some time after he could not walk with his head erect. Even now, after a lapse of twenty-three years, he cannot sit or stand for any time without feeling pain in his back between the shoulder blades. Five years ago he was thrown from a phaeton, and fell upon his head, fracturing both his clavicles. His habits are intemperate. He had never had rheumatism or syphilis, but has for the last six months been troubled with a cough and expectoration, which is mucus, containing blood. He has such an amount of dyspnoea on walking up a hill as frequently compels him to stop and rest.

On admission, his left pulse was found to be smaller than the right, 76 in a minute; his pupils both alike. The heart's apex beat between the fifth and sixth ribs, about half an inch inside the nipple. One inch to the left of the sternum, dullness extended from just above the fourth rib down to the liver



dulness. In the nipple line (along the fourth rib) dulness extended from the left edge of the sternum two inches to the left. The left side of the chest in the mammary region was somewhat more prominent than the right. The heart's sounds were normal both at apex and base. The second sound seemed to be somewhat accentuated in the right carotid, but decidedly so in the left close to the clavicle; and when the finger was pushed close into the tracheal fossa it came in contact with a distinctly bulging and pulsating tumour just under the sternal end of the left clavicle. In the right subclavian there was a slight systolic bruit, followed by the second sound. In the left subclavian there was no systolic bruit, but the accentuation of the second sound was well marked. The percussion and auscultation of the lungs was normal. The blood in the expectoration was fluid, arterial, not frothy, and small in quantity. *Diagnosis*.—A small weeping aneurism of the arch, implicating the immediate origin of the left carotid, and communicating by a minute opening with the left bronchus. *Treatment*—

R Potassii iodidi, ℥vj.

Infusi chiratae, ℥vj. Solve.

Sig., One tablespoonful three times a-day, rest in the recumbent posture, and ordinary full diet.

On 8th July it is noted that the bruit in the right carotid had quite disappeared, while the second sound in the left carotid did not seem to be so greatly accentuated; the spitting of blood had ceased. On the 9th of July, he spat up a few pellets stained with blood for the last time. After this he continued steadily to improve till his discharge on 8th November 1869, all the aneurismal symptoms being much ameliorated and the dyspnoea gone. During the summer of 1870 he was in another ward of the Infirmary, and I then ascertained that he had remained well, and that no trace of his aneurism could be detected. It is obvious that amidst all the difficulties which obscure the diagnosis of aneurism, especially one so uncertain as to its seat and so limited in its extent as this one was, this case must only be taken for what it is worth.

The symptoms were, however, so fairly well marked, and the relief from treatment so great, that I think I am justified in regarding it as proof of the efficacy of this treatment, even in aneurisms of this peculiar class.

I might easily multiply histories of aneurisms treated by the iodide of potassium, but I think a sufficient number has been given to illustrate the mode of treatment and the amount of relief to be attained. I have now treated after this fashion a very considerable number of cases of aneurism, of whom the most of those not related were clearly cases of aneurism of the aorta, mainly of its thoracic portion, and all with a similar result, viz., speedy relief to pain and suffering of every kind, while in a few there has been so complete a subsidence of the tumour and relief to all the symptoms, as to amount to an apparently perfect cure. These results are extremely encouraging, and when we reflect upon the entire absence of any risk to the patient from the treatment, and the almost absolute certainty of relief to his sufferings, and prolongation of his life being at least attained, I think I am warranted in saying, that no treatment for internal aneurism hitherto devised holds out anything like an equal prospect of relief, if not of cure, with that by the iodide of potassium.

In regard to the action of the iodide of potassium in aneurism, there is no reason to suppose that aneurism and syphilis are so invariably causally related, as to lead us to ascribe the remarkable curative properties of iodide of potash to its antisyphilitic virtues. While even if this were the case, it would only make its action more mysterious and less easily understood than we expect to be able to show it to be.

Dr Chuckerbutty, as I have already stated, supposed the iodide to increase the coagulability of the blood, and Dr Roberts seems to consider this a probable theory. The correctness of this idea is, however, doubtful; it might explain the coagulation of the blood in the aneurismal sac, but it could not explain the great relief to the pain which is the almost



previous consolidation had been due to solid coagula. After six years Wilson died and the mystery began to be solved. What seemed to be a firm solid nodule lying on the abdominal aorta, was found to be really an aneurismal sac containing nothing but *post-mortem* clots, its seeming solidity being due to the development of a calcareous plate in its anterior wall. Already, however, we had got the clue to what seems to be the true action of the iodide. From the first it had been noticed that under its influence the pulsations within the sac and throughout the arterial system were much diminished in force, and this had been so marked in the second case (Case XXXVII.) as to attract the attention of the patient himself, and had been rightly referred to reduction of the intra-arterial blood pressure. This, however, on the strength of Wilson living, had for six years been regarded as a mere adjuvant to blood coagulation, hitherto the only recognised method of cure. Wilson dead, however, told a different tale, and showed that lowering of the blood pressure in promoting the cure of aneurism did not so much aid blood coagulation, as simply sac contraction. An aneurism is formed because the normal blood pressure suffices to overcome the resistance of some weak part in an artery, which it gradually dilates with each pulsation till it ruptures. Lowering of the blood pressure might stop any further dilatation, but it could not promote a cure unless the sac were simultaneously filled with coagula, or its walls became thickened and contracted. *Post-mortem* examinations teach us that under the influence of iodide of potassium coagula are only occasional and concomitant, and that the essential relief is obtained by thickening and contraction of the wall of the sac. In one case already referred to (at p. 358) the iodide treatment had been followed by marked benefit, the superficial pulsation had almost completely disappeared, and there had been simultaneous diminution of all the pressure symptoms. The patient died in the wards of a colleague from a different disease. An aneurism the size of a small orange was found coming off from the ascending aorta,



and from this a smaller sac, the size of a walnut, projected anteriorly and was adherent to the thoracic wall in the third interspace. No trace of clot was found within the aneurism. Sections were made through various parts of the sac, and on examining them microscopically, the intima was found to be everywhere diseased, granular, degenerated, and irregular in its thickness as if being worn away. The muscular layer of the media was tolerably dense, apparently hypertrophied, over the large sac close to the artery; over the small sac, at its external part, it was still present but granular, and undergoing fatty degeneration. The elastic layer of the media did not appear to be atrophied or degenerated in any part examined. The adventitia was considerably hypertrophied, especially over the outer portion of the smaller sac.\* This hypertrophy of the muscular coat, where that still exists, and of the adventitia, with concomitant contraction, are found in all aneurisms which have been treated with the iodide with any measure of success. We may refer it if we will to some hitherto unsuspected action of the drug on the muscular and fibrous tissues, but it seems more rational to regard this hypertrophy of their muscular and fibrous tissues, under the conditions present, as but another manifestation of that well-known law (Paget) by which a hollow muscle hypertrophies when opposed to an obstacle with which it is able successfully to contend. This theory has also this further advantage, that it gives us a true physiological basis for our treatment, instead of a mere hypothetical therapeutical idea, and that when intelligently acted upon we more certainly and quickly attain that amount of success which is possible. In recent times, foreign pharmacologists, and chiefly a Russian experimentalist named Bogolepoff, have confirmed the views just stated, so far as to show that one of the chief actions of iodide of potassium is to lower the blood tension uniformly throughout the body, by dilating the arterioles, the heart's action being at the same time diminished in force. In dogs this diminution of blood

\* Case of Murray, *vide* p. 358; and *Ed. Med. Journ.*, June 1876, p. 1142.

pressure and of cardiac force is frequently accompanied by increased frequency of the heart's action, unless the dose is much increased when slowing of the heart's action occurs as a precursory stage to paralysis. In frogs, a large dose dilates the arterioles, and slows the heart without any precursory stage of rapid action.\* So far as the action on man is concerned, we may accept uniform dilatation of the capillaries as the cause of that lowering of the blood tension which is so important as a remedial agent in the treatment of aneurism. The action on the human heart is to diminish both the force and frequency of the pulsations, and this occasionally in a very marked manner. More often, perhaps, in the doses in which it has been hitherto prescribed for aneurism, there is a comparatively unimportant rise in the frequency of the pulsations, which appear not materially to interfere with the ultimate improvement of the case, though it probably prolongs it. But in some few the action of the iodide is more injurious, and the heart's action becomes unduly quickened up even to 170 beats per minute; this rapid pulsation has always a bad effect on the aneurism, and none of those in which it occurred did well. Having thus worked out the problem so far as to have got a distinct idea of what was wanted, lowering of the blood tension without increase in the number of the heart's pulsations, which seemed to be injurious, *non vi sed sæpe cadendo*, it remained to be ascertained whether acting on those principles would give us better, or more definite results, than the haphazard method hitherto pursued. Accordingly, those cases which came next under treatment were put to bed for a few days without further treatment, their pulse rate being carefully taken night and morning. So soon as the average pulse rate in recumbency had been sufficiently ascertained, ten grains of iodide of potassium in some bitter infusion, usually chiretta, were given three times a-day. If the pulse rate

\* "Zur Frage der physiologischen Wirkung des Iodkalium," *Moskauer pharmacology. Arbeiten*, s. 125; and Virchow's *Jahresbericht*, 1876, s. 402.



remained unchanged, the dose was increased to fifteen grains three times a-day, and we have not yet been able to get beyond this dose; while very often we have not been able to give more than ten grains without raising the pulse rate. When the iodide has been thus administered, the success attained has been quite remarkable, the cessation of pain, the diminution of pulsation, and the general improvement of the cases have been so marked, and so rapid, as to satisfy me that we have now got the clue to their proper treatment. It is unnecessary to give any cases in detail; there are constantly cases in the wards undergoing treatment, and it is sufficient to say that while formerly, with large doses, six months was the average time before marked improvement was obtained, we now obtain similar results in three months and sometimes in less. Of course we are not more successful in curing our patients than formerly, and they still require to be more or less under treatment during all their lives, taking the iodide at intervals. But I think we are now entitled to say that the amount of relief obtained is no longer a matter of chance, but is largely proportionate to the period at which we begin our treatment. So that if we get the patient early enough while there is yet but little change in the media, the relief is rapid, and the cure may be almost complete, while if we have nothing but the adventitia to rely upon, a longer treatment is required, and the relief is less complete, though even in cases apparently of this character the improvement is sometimes very remarkable.\* I have only twice seen cases of complete intolerance of the iodide, in one case even the smallest dose gave rise to rapid pulse and to severe neuralgic pains of the abdomen, even when the patient did not know he was taking it. In the other, its use was so constantly followed by copious herpetic eruption—so-called hydroa—that it had to

\* This doctrine as to the action of iodide of potassium in aneurism will be found enunciated in the *Ed. Med. Journ.* for June 1876, at p. 1142, and in the *British Medical Journal* for April 5, 1879, at p. 511, forming part of the reported discussion as to the treatment of internal aneurisms at the Glasgow Pathological and Clinical Society on March 5, 1879.



be given up. Iodide of sodium had the same effect. This was the more to be regretted, as in both cases its action on the disease was markedly ameliorative.

In regard to the adjuvant treatment, there are one or two remarks which seem to me of considerable importance, and these may be comprised under the two heads of position and diet. Whatever is capable of lessening the frequency of the heart's action, without impairing the strength of the patient, or vitiating the quality of his blood, cannot but be an important adjuvant in the treatment of aneurism. The enforcement of the recumbent position, therefore, which is so influential in this respect, has seemed to me a matter of paramount necessity, and has been strictly carried out in almost all of my cases; indeed in the most serious one, the patient was laid upon his back for fully ten of the eleven months he was in my ward, not being permitted even to turn upon his side—any attempt to do so being always attended by a recurrence of his disagreeable symptoms. I have no doubt that the success attained in his case, as well as in others, was very considerably due to the long-continued perfect rest in the recumbent position; and in the treatment of so serious a disease as internal aneurism, I should consider it most unwise to neglect the employment of this simple but efficient mode of aiding the cure. No doubt the enforcement of this portion of the treatment is irksome and impossible of attainment without the intelligent acquiescence of the patient,\* but I have not had any difficulty in obtaining this, on explaining my reasons and the object I had in view; and I may make the same remark as to diet. Aware of the evils of starvation on the one hand, and of plethora on the other, my patients were at first put upon a somewhat restricted mixed diet—fish, chicken, or rabbit being given for dinner, at first at all events, more easily

\* Great relief, however, is often obtained from this treatment without rest in the recumbent posture, and where it is desirable to prevent the patient from knowing his own condition this may be dispensed with; but I would not willingly do so in any serious case, and what case of aneurism is not serious?

digested generally and less apt to give rise to so-called biliousness than beef or mutton, &c., while they were told voluntarily to restrain their appetite as much as possible, and to make use of no more than what they felt to be sufficient to maintain themselves. In regard to fluids, water, tea, or milk alone were allowed; and though they were not doled out in a measured quantity, yet similar directions were given in regard to them as in regard to solid food; explanations were given in regard to the result desired, and the evils to be avoided, and careful inquiries were daily made as to the mode in which this advice was being complied with, so as to impress its necessity and importance upon the patients. I have had no reason to be dissatisfied with the result of this reciprocal confidence between patient and physician, and I believe it to be more conducive to the well-being and the comfort of the former, than any more precise definition of amounts by weights and measures. The principles of the treatment were intelligibly laid down, and intelligently acted upon, and the effects daily noted; and the result has been, that all of my patients have, without becoming plethoric, yet preserved a healthy and well-nourished appearance throughout the whole of their tedious treatment. Latterly, however, I have been less scrupulous in this respect, having had even better results when full diet was allowed;\* and while the unnecessary ingestion of fluids should be avoided, the iodide produces such free diuresis that this requires the less to be insisted on. Alcohol, however, in any form, I have found to be certainly injurious, and its use should never be permitted, except when it is absolutely needful, and then only temporarily.

I need hardly say that, notwithstanding the great and manifest relief obtained in almost all cases of aneurism by this treatment, positive cures can only be exceptional, of comparatively rare occurrence, and the result of long-continued and careful treatment. An aneurism may prove fatal in many various

\* The iodide of potash is believed by some (*vide* Kämmerer, Virchow's *Archiv.*, bd. lix.) to destroy the albuminates in the blood, and for this reason we must see that our patients are sufficiently fed.



ways besides rupture, and the relief obtained can only be regarded as unequivocally tending towards a cure, when, from the symptoms, it is apparent that the aneurism is shrinking in all its dimensions. Even then incautious exertion may rupture the sac at some weak point, and death may thus ensue while the patient is flattering himself that he is being cured. Compression of important organs by a solidified sac may induce dangerous and even fatal complications, and even under the most favourable circumstances, the occurrence of an aneurism of a large and important artery proves the existence of such a diseased condition of the arterial coats as will render the patient's life ever after a precarious one.

Notwithstanding the great relief to the symptoms in almost every case, it is obvious that anything beyond this—anything at all approaching to a cure—can only be expected when the aneurism is detected early, and chiefly in comparatively young individuals, in whom there is probably a less seriously or generally diseased condition of the arterial coats than in those who are more aged. On the other hand, if there is any evidence of a greatly diseased condition of the arterial coats, this would form an unfavourable element in estimating the probability or completeness of a cure; and the presence of more than one aneurism in the same person would seem likely to reduce still further the chances of anything beyond mere temporary improvement. But the history of Case XXXVIII. shows that even in such cases this improvement may be so great as to be well worth all the trouble expended in attaining it.

The results obtained were unquestionably due to the iodide alone, though adjuvant treatment no doubt assisted materially in bringing them about. Still the iodide alone is quite capable of producing by itself a most marked benefit. In 1873, I had under my care a gentleman, with a large aneurism of the aortic arch; his main symptoms were a remarkable *vox anserina*, and constant headache, flushed and turgid face, &c., the result of compression of the large veins. Under the use of the iodide in twenty-grain doses, without any other treatment, all these



symptoms were so greatly relieved, and even his voice so much improved, that he ceased to think himself an invalid. This gentleman subsequently relapsed from over exertion, his head and arms were so turgid, his quasi-apoplectic symptoms so severe, and his aneurism so large, that a late distinguished physician gave him only a fortnight to live. Nevertheless he recovered so completely that the last time I saw him it was to be reproached for my mistaken diagnosis, as some medical friends had told him he had no aneurism. I need not add that I could still detect the aneurism, and told him so; he died last year from embolism. You will distinctly remember then, that while I do not claim that we can perfectly cure aneurism by iodide of potassium, or by anything else, yet I am quite certain that at the present day we possess no other remedial agent or mode of treatment which so surely gives relief, and so frequently prolongs life, as the iodide of potassium; even apparently the most hopeless cases being not always beyond its palliative influence. While therefore I freely acknowledge the importance of recumbence as insisted upon by Tufnell, and even admit the possible utility of a limitation of the diet in such cases—within reasonable bounds—I claim for the treatment by iodide of potassium that its action is in strict accordance with physiological laws, and that it is capable of relieving the symptoms of aneurism more uniformly and effectually than treatment of any other character, and that even without any adjuvant treatment whatever, though, of course, its success is most materially promoted by such treatment.



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